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Original Articles.

THE PHILOSOPHICAL RELATIONS OF NEUROLOGY.

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ALL special sciences, the positive as well as the practical, naturally seek incorporation into some one comprehensive scheme of thought which embodies man's largest, and at the same time most centralised, conception of the Whole of things, which is his Philosophy or Rationale of the Universe. The striving after some such organic unity in knowledge seems to be an universal law of human intelligence. The attempt to grasp it prematurely, so introducing an illusory unity in place of the real one, has been the source both of much hasty scientific generalisation, and of much obstinacy in cleaving to antiquated theories in the face of newly-discovered or unfamiliar facts. But in itself the striving is so far from being irrational, that it is difficult to imagine any intelligence which it does not in some measure dominate.

We see the effect of this tendency not only in the coordination and subordination of special departments under some single comprehensive science—neurology, for instance, under biology—but also in the conception of the greater and more comprehensive sciences themselves, divided into the two groups of positive and practical, depending, so far as their cardinal or axiomatic conceptions are concerned, upon the philosophical analysis of consciousness, as the original charter of their validity. This is true both of what we may call the great ultimate positive sciences, mathematic, kinematic, dynamic, physics, chemistry, biology, and psychology, and also of the great ultimate practical sciences, namely, logic, æsthetic, ethic, and theology. In all these sciences alike, the fundamental ideas and conceptions, and therefore also the hypotheses built upon these, must be such as to endure the scrutiny of an analysis which separates what is permanent and self-evident in all experience from assumptions which owe their seeming necessity only to long association and familiarity.

From this it follows that the organic unity of man's knowledge as a vast whole must be established, if at all, by philosophy. But philosophy would be wholly unable to respond to the demand if it was not in the possession of a field and of a method peculiar to itself, which guarantee it an existence independent of the several sciences which it brings together, and constitute it a special mode of knowledge distinguished from science, and yet directed to discover truth of fact, not merely to effect a convenient arrangement of facts discovered in other fields and by other methods. In the latter case philosophy would itself be nothing more than a luxury or convenience, varying with the purposes which might happen to be entertained by the individuals pursuing it.

Now the peculiar field of philosophy as contradistinguished from science, a field which is special to it as philosophy and yet common and universal, inasmuch as co-extensive with the ground covered by the sciences taken together, is consciousness; that is to say, the knowing of existence contrasted and connected with its objective aspect, namely, with things, persons, and their functions, taken as existents already known, and appearing to be given immediately to perception. These latter are the form of experience with which the sciences deal. Philosophy alone deals with the knowing or experience out of which that form of experience arises which is the knowledge of these objects. Knowing as distinguished from Being, and yet connected with Being as its object, is thus the field of philosophy—a field which on this account is at once peculiar to itself and yet common to all the sciences. For in marking it out philosophy bases itself upon a distinction, within experience, which no other science draws. And its field being thus marked out, the method which it pursues therein follows from again applying the same distinction. It is the method of subjective analysis of consciousness, watching for assumptions and excluding them from the number of ultimate elements of knowing, which it seeks to discover.

The field of philosophy—existence on its subjective side, or consciousness, contrasted and connected with existent objects; the method of philosophy—analysis of that consciousness without assumptions; these are the properties by which philosophy is constituted as an independent mode of knowledge, enabling it to comprehend in a single view the objects of the rest, and to do this by simple adherence to the truth of immediately known fact, without any arbitrary adoption of principle, or favourite purpose to be served by the unification.

At the same time no other mode can transcend or be more comprehensive than this. Knowing and Being are complementaries of each other, because consciousness is the point of view which all knowledge necessarily occupies. There may be Being which is not endowed with consciousness, but there can be no Being of which we are wholly unconscious, that is, which we do not think of as Being in naming it, even though our next step may be to deny its existence in the shape under which we thought of it. All such distinctions, therefore, as that between things as they are and appearances are minor distinctions falling within this most comprehensive one. Consciousness embraces all Being, and there is nothing beyond consciousness with which it can be compared or by which it can in any way be limited.

So much with respect to philosophy it has been necessary to premise, in order to show the sense in which neurology (or anything else) can be said to stand in relation to it, neurology being taken as a special department of the comprehensive science of biology. Now nerve substance, the organisation and functions of which are the objects of neurology, taken simply as a highly specialised form of matter, stands in a very remote relation to philosophy. True, philosophy has to account for the idea of matter, and for the belief in a material world, out of the analysis of consciousness spoken of above, and to show the grounds for and against the belief in its independent reality. But this being done, the function of philosophy in that direction is at an end. The laws of matter and of all the special forms of it belong not to philosophy but to science, whatever may be held concerning its character as a real existent. And in this respect philosophy has nothing to do with any form or mode of matter so highly specialised as nerve substance.

The more specific relation, which as we shall see is a very close one, between neurology as the science of nerve substance and philosophy must therefore be sought in quite a different direction, at the upper not the lower end, so to speak, of the series of positive sciences. Neurology examines that most complex and highly organised structure in living beings, upon which consciousness depends, in all forms and all degrees of it, from simple sentience and from surmised latency, upwards to the most complex phenomena of thought, emotion, and volition, and to the most distinct awareness possible to self-consciousness. Nerve substance and neural phenomena are that highest portion of biology in which the domains of biology and psychology overlap each other, and in which all psychological explanations must ultimately be sought.

The domains of biology and chemistry overlap each other in one direction, as those of biology and psychology overlap each other in another. The same substance is both chemical in composition and living in operation. And again, the same substance is both living in operation and conscious in function. Whereby I do not mean to affix any specially technical sense to the terms, *composition*, *operation*, and *function*, but use them simply in order to indicate the phenomena intended with brevity. The same phenomenon of overlapping prevails throughout the whole series of sciences including philosophy, just as a similar phenomenon is observable in the substances and forces of nature, and in the states and processes which compose the varied stream of consciousness as it comes to us in actual experience. The connection then between neurology and psychology is of the closest. The states and processes of consciousness one and all depend immediately upon states and processes of nerve substance, or at any rate there is nothing to indicate the presence of any intermediary. The two kinds of phenomena are essentially different, one material, the other immaterial, and the latter are dependent concomitants of the former.

But it will be said: How does this close relation between neurology and psychology affect the question of this paper, namely, the relation between neurology and philosophy? Or rather it may be asked: How is it compatible with the view of philosophy taken above, seeing that philosophy was there represented as having a position wholly independent of science? Consciousness, it may be said, was there held to be the special field of philosophy; and here it is admitted to be dependent on neural phenomena, the object of a special science. If consciousness be dependent on the functioning of nerve substance, must not philosophy be dependent on the results of neurology? If so, what becomes of its special field and independent position?

It is this very point which the present paper is intended to elucidate. We find as a rule those psychologists who have no definite philosophy, or who have only a theological one, or who think that over and above science there is nothing but religion, which is a matter solely between a man and his God—we find, I say, psychologists of these varieties very reluctant to admit as a fact the complete dependence of consciousness on nerve process. Concomitance they will admit, and even reciprocal dependence. But dependence of consciousness on nerve process simply, without dependence of nerve process on consciousness, this they hesitate to affirm until the latter dependence shall be disproved in detail, unless indeed their interest goes in no respect beyond an interest in science. In all these cases, psychology—the last and highest of the positive sciences—does duty for philosophy; so that in them to be materialist in psychology means being materialist in the fullest possible sense of the word. Hence the importance of the point now under consideration for all those who admit the complete dependence of consciousness on nerve process, and at the same time wish to take such a general view of the knowable universe as may properly be entitled philosophical.

Turning then to the point under consideration, the apparent difficulty which it raises is surmounted by showing that the domains of psychology and philosophy overlap each other, one portion of each being common to both, just as in the cases of chemistry and biology, biology and psychology. The common ground between biology and psychology is covered by neurology, the science of that kind of living matter which has consciousness immediately depending on it. And psychology has necessarily to take account of the phenomena of neurology, because they are the immediate real conditions upon which consciousness depends, and without reference to which no explanation whatever of the origin and concatenation of process-contents of consciousness would be forthcoming. The study of this immediate relation between nerve process and consciousness is properly designated as psycho-physic. And what remains peculiar to psychology, over and above the psycho-physical portion which it shares with neurology, is the discrimination and study of the various processes of consciousness, both severally and in combination with each other, such as sensation, perception, attention, memory, recollection, imagination, appetition, thought, emotion, volition, and so on—of everything, in short, which used to come under the head of faculties, recently christened functions, of the mind.

Similarly the domains of psychology and philosophy overlap each other, but here with a difference arising from the peculiar nature of the ground which they share in common, and which, as already said, is consciousness in its totality. The difficulty which is here raised, and which we have now to surmount, is this. The whole of consciousness is common to both, and thus *prima facie* what has been called overlapping, becomes co-incidence. *Prima facie* there is room for one science only, not for the science of psychology and philosophy as well, unless we identify the one with the other. The question is, how is it possible to establish the justice of the term *overlapping* as applied to this instance? Or in other words, how is it possible to maintain the distinctness and independence of psychology and philosophy, while admitting that the phenomena which they cover, the phenomena of consciousness, are, in their whole extent, common to both?

The reply comes from the side of philosophy. It is given by that distinction which, as was said above, philosophy alone draws, and the truth of which is the title-deed of its validity, the distinction within experience of Knowing and Being. Every state or (better) process-content of consciousness has a double aspect. In one aspect it forms part of what we may figuratively call our subjective panorama of the existent world; in the other it comes into and passes out of existence, as itself an existent for a brief period, in dependence on the nerve process which subserves it. Just as the generations of mankind are born into life and pass away in death, so the successive layers of consciousness rise into being and pass away into nothingness, never to return. The *layers* must of course be understood as sections, artificially distinguished by subsequent observation, in what we may figuratively call the time-stream of consciousness, flowing not from point to point in space, but from present to past in time only. Thus, for instance, our perception of a single fixed object in space, say a picture on the wall, looked at for five minutes, consists really of a succession of etherial vibrations beating on the retina and accompanied by sensation, those vibrations which occurred at the beginning of the five minutes having ceased to exist and condition sensations, when those at the end of the five minutes are occurring.

Every kind of process-content of consciousness is alike in this feature of successiveness; not only sensations of external sense, but all sensations, emotions, images, memories, associations, recollections, conceptions, attention, sense of effort, thought, reasoning, comparison, conscious volition, choice, and resolve, sense of beauty and deformity, of right and wrong, of pleasure and pain, in all their countless qualitative and quantitative varieties. And yet all alike, being in the usual phrase recalled or recallable in memory (though, strictly speaking, a recall of one and the same state of consciousness is impossible) go to form part of that subjective panorama which is our knowledge of the existent or surmised world, and thus together make up that aspect of consciousness which we call knowing. All alike owe their genesis, the order in which they actually occur at first, and also the order in which they, or rather their similars, are finally disposed, before the panorama which they constitute becomes an intelligible picture, or deserves the name of knowledge in the full sense, to the nerve processes which subserve them, and which operate again and again in similar ways on the occurrence of similar stimuli.

As the conscious life of an individual proceeds from infancy onwards, his consciousness is thus continually occurring in the form of a time-stream of many currents, each current consisting of successive changes in consciousness, thrown up and determined by nerve processes, of which he is wholly unaware, unless and until he chance, in later life, to become acquainted with physiological results. This aspect or feature in consciousness—I mean its dependent aspect of genesis and determination by nerve process—is what may be called consciousness as an Existent, and not as an Existent merely, but also as a particular and individual Existent, in spite of the universality which it possesses in its other aspect or character of a Knowing. In its character of an Existent it is attached to and dependent on some particular and individual living body, one among many, each of which may similarly be inferred to be the seat of a similar individual consciousness.

It is this existent aspect of consciousness which is the object of psychology, since psychology as a positive science has necessarily to busy itself with the laws of origin and history of the phenomena which are its special field. Psychology, therefore, has not to account for there being such a *nature* as consciousness, for this is the primary *datum* of all particular knowledge whatever, but, taking this nature as already known, to account for the genesis and subsequent history of it in individual living beings, that is, in the form of individual consciousnesses. And in order to do this it is obvious, that psychology must also treat the nervous system and process as realities or real existents, no less than consciousness; and yet our whole knowledge of these is part of, and bound up with, our knowledge of the whole material world, into the real existence of which it is beyond the scope of psychology, as a special science, to enquire. This it has to assume as already known, and known as different from the consciousness which it is called in to account for.

A similar remark holds good in the case of those psychologists who account for the genesis and history of consciousness by the hypothesis of an immaterial agent, be it Mind, Soul, or Ego, and whether alone or in conjunction with a nervous system. I mean that they, too, must assume the existence of such an agent as a reality, and also, as in the former case, a reality known as different from the consciousness of which it is supposed to be the Subject.

Thus, whatever be the type of hypothesis adopted by psychology, it is plain that a real existent or existents, of some kind or other, must be hypothetically assumed, in order to account for that other existent, consciousness, which is the object of the science. Whence can the knowledge of such hypothetical existents be derived? Whence the knowledge that consciousness is an existent? Whence the knowledge of the meaning of the term *existence*, or of the very ideas of genesis, of history, of dependence, of explanation by reference to real causes or conditions? It is evident that all this knowledge can be derived only from the other aspect of consciousness, its aspect as a knowing or subjective panorama of existence, some parts or features of which must be prior to, and independent of, other more particular ideas or more particular knowledge, which are subsequently developed in or incorporated with it. For it is plain that the panorama cannot, simply as such, be credited with a power to make assumptions, as if it were an individual person speculating on data already given to it. But this half or aspect of consciousness is that which we have seen that philosophy, which draws the distinction originally, claims for its special province, as distinguished from that of psychology, which is a positive science. And it is also evident that this province is something more than a merely co-equal aspect or half of consciousness as a whole. The knowledge of which it consists is not a knowledge of its own province only; it is a knowledge of its own province as contrasted with that of psychology; a knowledge which, therefore, embraces both provinces and the nature of the distinction between them. Hence it is that philosophy, besides being the more specialised of the two, is also the more comprehensive in its grasp of the phenomena common to both. This is a relation which may be illustrated by what has often been remarked about space, I mean, that a knowledge of what is called its third dimension alone enables us to understand its second, or plane superficiality, by furnishing a direction with which on the ascending side it is in contrast.

The distinction between Knowing and Being, upon which philosophy bases itself immediately, and upon which it shows that psychology is based mediately, that is, by intervention of the idea of real condition, which is itself derived from that of real existent, is a necessary and inevitable one. I hope I have gone some way towards showing that it is so. To go farther would involve me too much in the analysis of consciousness as a knowing, which would be an unfit topic for the present occasion. The same must also be said of any attempt to show how the knowledge of an external material world arises from the time-stream of consciousness, which, when spatially extended perceptions, visual perceptions for instance, occur in it, becomes a panorama, and which in relation to that real material world we may call subjective.

The relation which I wish to establish between neurology and philosophy has psychology as its intermediary link. Nerve process conditions consciousness as an existent, hence psychology overlaps neurology. Consciousness as an existent in dependence on nerve process is only known through consciousness as a knowing, or subjective panorama of existence generally, hence philosophy overlaps psychology. Assuming, as I have here done, what seems to me the only working and workable hypothesis possible, namely, that consciousness as an existent depends on nerve process, and not reciprocally, the remaining difficulty is that which I have tried to overcome by showing that consciousness really has, and necessarily has, the two aspects distinguished, but not separated, by the line drawn as I have drawn it between Knowing and Being. It is a line which passes, to speak figuratively, right through the middle of every state or process-content of consciousness, whether simple or complex. And on this line of demarcation the whole of philosophical speculation may be said to depend.

Take a simple illustration of its nature and necessity. Suppose a candle to be set up in a dark room and subsequently lighted, and let the light of that candle represent consciousness. The light has a double aspect, analogous to that of consciousness. It illumines the room and the objects in it, including the candle and the candle flame, and is itself visible by its own light. Without it nothing of all this would be visible at all. Here it corresponds to consciousness in its aspect of a Knowing. But on the other hand, without the room and the candle, and the match applied to its wick, the light itself would not exist, and it exists no longer than for the interval from the applying of the match to the burning out of the candle, upon which it immediately depends. Here is the same light in its character of a real existent. The theory of the composition of the candle, the ignition, and the gradual burning, is the neurology and psychology of the light.

Thus the whole mechanism of consciousness considered as an existent belongs to psychology, and the whole content of consciousness considered as a knowledge of existence belongs to philosophy. And if any one should be inclined to suppose that this is a mere contention about words, and that it is really indifferent whether we call either domain or both by either name, the reply is, that the names are important because, and only because, they mark an essential difference, which cannot be ignored, between two kinds of problems and two kinds of method, a difference therefore which it is indispensable to keep well defined for the sake of clear and consistent thinking.

It is impossible to treat the mechanism of consciousness clearly and satisfactorily, without first framing some working hypothesis of the Subject or Agent of that mechanism. And every such hypothesis must either be drawn from the crude assumptions of traditional Animism, or else from the knowledge of real existents, guaranteed by a careful analysis of the phenomena of consciousness without assumptions; that is, of the phenomena considered, in the first instance, in purposed abstraction from hypotheses of every kind concerning their genesis. To begin by laying it down that man consists of mind and body, or that knowledge depends on the presence of Objects to a Subject, is to begin with assuming the truth of the very conceptions which science has to investigate and explain, and the adoption of which, as the initial conceptions of an explanatory science, has already entangled psychology in a labyrinth of confusion. For thenceforward it is necessarily occupied, not as it should be with explaining the phenomena of consciousness, their order and history, by connecting them with the hypothesis adopted, but with explaining in what sense the terms *Mind* and *Subject* are *not* to be understood, if they are to be compatible with the phenomena at all. It is occupied, in short, with endeavouring to explain away, or avoid the necessity of employing, the very assumptions which it has made as the basis of an explanation. Common sense tells us that there are phenomena which we ordinarily use these expressions to describe. The question for psychology is, what are the real (but always phenomenal) facts and laws, of which these expressions are the familiar description.

But there is another consideration which exhibits in even a stronger light the confusion of thought which inevitably results from treating the content and the genesis of consciousness, together and undistinguished, as the object-matter of a single science, whether that science be called psychology or philosophy. Consciousness taken in its content, or as a Knowing, is a knowledge of existence generally; all things are its object. But consciousness taken in its genesis and history, or as an Existent, is an existent of one particular kind only; material things and the physical forces of Nature being existents of other kinds. Every single person's consciousness has both these characters. And if they are confused, every single person must logically hold the universe to be his own creation, since he must at once generalise his individual agency and individualise his general knowledge. My point is, not that this position is absurd, though it undoubtedly is so, but that between such a psychology (or philosophy) as this and other sciences, no possible congruence can be brought about.

The true distinction, therefore, upon which all possible harmony depends, whether between the sciences themselves, or between them taken collectively, and that philosophical branch of knowledge which unifies them, is the distinction between consciousness as a content of experience and consciousness in its genesis as an existent, which then gives rise to the further distinction between consciousness as an existent and its real conditions, as the primary distinction between philosophy and psychology. This, I repeat, is not the distinction between Mind and Body, nor that between Subject and Object. These latter must be conceived as existents only, real or imagined, and belong but to one half, one member, of the true distinction between consciousness and existents generally, consciousness itself having also a distinct character as an existent. The true ultimate distinction falls within consciousness itself. And on this basis all the other sciences find their place and their validity on exactly the same footing as psychology itself finds it, when treating of consciousness as an existent.

Mind and nerve substance are rival claimants for the office of being the proximate real condition of consciousness. The question is, What is known about their reality and respective fitness for the office claimed? Of mind I venture to say nothing whatever is known, neither its nature nor its reality. Consequently it can have no fitness for the office at all. It is a fiction of the fancy, tracing its pedigree to the animistic theories of pre-historic times, before anything was known of the more recondite properties of nerve and brain. Analyse the content of consciousness, the subjective panorama of existence, in which the title-deeds of all our knowledge are to be found, and nowhere will you find evidence of any entity or agency answering to the terms *mind* and *mental, psyche* and *psychical*. Everywhere there is consciousness, and everywhere there is evidence of nerve substance and neural agency supporting consciousness.

But it is not this which I am writing to tell the readers of BRAIN. The point which I wish to enforce is, that with a psychology such as I have criticised, a psychology based on the immaterial entity Mind, neurology is cut off from any direct connection with philosophy. For then nerve substance is no longer conceived as the proximate real condition of consciousness, but an immaterial, and, therefore, totally heterogeneous substance is introduced, which cannot be brought into intelligible relation either with consciousness or with nerve, seeing that there is nothing definite or positive about it. The real and direct connection between nerve and consciousness is severed, and nothing remains but the ideal and indirect one through matter generally, matter itself being regarded as a creation, possibly illusory, of the mental powers. Psychology, pursued as it is at present pursued by the greater number of its most approved professors in this country, is not only a heterogeneous member in the series of the sciences, but also tends, from the nature of the entity which it assumes as its basis, to swallow up all other sciences, and exhibit them as subordinate departments of itself. For if everything material is the creation of mind and has no other than an ideal existence, the nature and laws of everything material can only be understood, in the last resort, by referring them to the nature and laws of mind, that is, by the science of psychology.

Before concluding I would recur once more to the primary distinction between Knowing and Being, which alone enables us to bring the several sciences into a single unified scheme. I do so because the distinction is not one that lies on the surface, and it is therefore worth while to mention the special characteristic which enables it to serve as the basis of philosophy. The essential point in that distinction, as opposed to the distinctions between Mind and

Body, or Subject and Object, is this, that the Knowing includes no real agency, though it is open to proof of involving it if such proof should be at any time forthcoming, whereas Mind and Subject, both of them, imply real agency, inherent either in consciousness itself or in the being who possesses it. This is an assumption which as an initial assumption is illicit, since it is not a self-evident fact; and whoever is permitted to make it unchallenged may with equal justice demand, that its special nature shall also be assumed on the sole warrant of his assertion; whereby the door is opened to fictions without end.

It is understood and admitted on all hands that Being or Existence, to be recognised at all, must in some way or other be present in consciousness, must be an object of knowing in some general sense. But this does not require that the consciousness or the knowing should itself be known as an existent or as having agency in it or at back of it. In other words, it is consciousness simply as knowing which is the first and only strictly necessary implication in all knowledge of Being or Existence. That alone is the source whence all our knowledge of Being, Existence, Agency, Genesis, and so on, is derived. It is true that, when we have derived this knowledge from it, we see that consciousness must itself exist, and therefore have some agency either in it or at back of it, in order to be a Knowing at all. But this knowledge is subsequent to the knowledge which is a process-content of consciousness simply, not given in and with the primary knowing, since it pre-supposes repeated comparisons between many different parts or contents of it. And from comparison is also derived the still more particular knowledge, what kinds of phenomena are found capable of standing in the relation of real conditions to others. Hence arises the well-known philosophical axiom, that the order of knowledge and the order of existence are opposite in direction, or, as it is sometimes loosely stated, what is first in order of existence may be last in order of knowledge, and *vice versâ*.

The particular knowledge of the material world, of physical agencies in it, of nerve substance and agency, and of their close relation to consciousness taken as an existent, are also derivatives of comparison, and are facts of experience having positive evidence in their favour. Immaterial substances, on the contrary, and agencies inherent in them, and agencies inherent in consciousness itself, either as an existent or as a knowing, are things which, so far as I know, have no positive evidence in their favour, are conceptions derived from early forms of thought and speculation handed down by tradition, and can now be retained in science and in philosophy only on the (assumed) footing of self-evident factors in primary experience.

The psychologists who retain these ideas seem to me purposely to avoid coming face to face with the fundamental question in psychology, as it is in every positive science, what is the nature, and where the seat, of the agency proximately concerned in the phenomena which belong to the science, and the laws of which it is the purpose of the science to discover. They adopt, not a working hypothesis which can be brought into explanatory connection with the phenomena, but some words or phrases which will enable them to go on writing and lecturing on the phenomena without one; which is a very different thing, and one which never carries them beyond the descriptive department of the science. In short, the Mind of which they speak is not a *vera causa*, though they use it as one; while at the same time they persistently avoid coming to any explanation on the point.

The English distaste for anything speculative or metaphysical assists them considerably in this procedure. The analysis of consciousness simply as a knowing, and the connection of consciousness with nerve substance as its only positively known Subject, are two lines of thought which are natural allies and closely inter-dependent. But the alliance between them will remain *in posse*, so long as the present school of psychologists can continue to persuade men of science, and especially neurologists, that the proper business of philosophy is, not to analyse consciousness, but to enquire (*inter alia*) into the transcendental nature of Mind and Mental Energy, while adopting them as phenomenal realities on the assurance of the psychologists. This remarkable mis-representation of the sphere of philosophy depends partly on supposing that there can be any reality which is not phenomenal (a supposition due to adopting the distinction between appearance and reality as primary and self-evident), and partly on confusing phenomenal realities with the crude conceptions of common sense. It is the psychologist's own business to see that he avoids incorporating the latter into his scientific theory. He cannot be permitted to give them rank as scientific hypotheses, and then call upon philosophy to vindicate their reality.

ON THE NATURE AND RELATIONS OF MIND AND BRAIN.[1]

BY J. S. BRISTOWE, M.D., LL.D., F.R.S.

Gentlemen,—I esteem it a very high honour to have been elected to the post of President of the Neurological Society, but a very great responsibility to have to deliver an inaugural address. And I need scarcely assure you that I have given anxious thought to the choice of a subject suitable for the occasion, and yet within my capacity to deal with, if not adequately, at any rate without serious discredit.

Of course a wide discretion is, under such circumstances, always allowed to the lecturer. At the same time it is naturally expected that his address shall have some relation to the objects or to the performances of the Society over which he is called upon to preside. Our Society is not simply an Association of physicians engaged in the study and treatment of diseases of the nervous system, but it includes among its members physiologists, psychologists, and metaphysicians; and hence it takes cognisance not only of the pathology of the nervous system, but also of its physiology, and of metaphysics.

Feeling myself, therefore, free to choose from among this wide range of subjects, I naturally discarded topic after topic with which I was fairly familiar, until finally, with the presumptuous recklessness of ignorance, I decided to plunge into the wilderness of Metaphysics, a territory which I have never yet attempted to explore, and of which I know nothing.

I daresay most of my auditors have, like myself, speculated from time to time on abstruse metaphysical problems which have presented themselves to the mind, without taking the trouble to ascertain what has already been done in such matters, or to bestow upon them serious and continuous thought; so as, on the one hand, to save themselves fruitless trouble, or, on the other hand, to weed their vague fancies, and collect and fashion what remains into some hypothesis or theory admitting of clear expression in language.

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[1] Presidential Address, delivered on January 22nd, 1891.

The question I propose to discuss has long been in my mind in a fitful kind of way, but I have not hitherto attempted to reason it out clearly, or to formulate any answer to it. It has reference to the nature and relations of mind and brain.

In discussing this theme I propose to begin from the beginning, and therefore to make a few preliminary remarks about space and time, matter and force: about matter and force because they are directly involved in my subject and are inseparable from it; about space and time because of their fundamental relations to matter and force, and because, in point of fact, it was while speculating concerning them that I drifted into the line of thought which runs through my address of this evening.

We are, and we live, in space and time. What then is space, and what is time? Space means simply the relative sizes or measurements of material bodies, and of the intervals which separate them from one another. Time means simply the relative measures of movement or change. Space apart from matter has no existence and no meaning. It is simply infinite nothingness. Time apart from movement or change has no existence and no meaning. It is simply eternal nothingness. Space, therefore, is related more especially to the passive or statical condition of things; time is related to their active or dynamical condition. Space implies matter; time implies force.

It is needless, perhaps, to add that we have special relations to space and time, in the fact that we have material bodies, which are acted on not only by external forces, such as that of gravity, but also by forces within, which induce constant molecular and other changes.

Elementary matter is held by chemists and physicists to consist of minute molecules, or atoms, which are indivisible, unchangeable, and indestructible, and are separated from one another by intervals which vary in size according to circumstances. It is also generally held, that the different so-called "elementary" bodies consist of specific molecules, which are uniform for each element, but which differ in size or weight, or both, and probably in form, for the different elements; that all complex bodies (bodies comprising multiple atoms of any one element, or associated atoms of two or more elements) are composed of groups of molecules combined, so to speak, into atomic aggregates, which, under special conditions and with certain limitations, act in their relations to one another and to other elementary atoms as do the simple elementary atoms themselves. The main grounds for this belief are: first, that all elementary and other bodies, which blend with one another chemically, unite in certain definite proportions, or multiples of such proportions; second, that when complex bodies are resolved into their elements, these present absolutely the same characters as always belong to them in their free state; and, third, that simple elementary matter, and even many compound bodies, may exist under various conditions, such as the solid, the fluid and the gaseous, and thus occupy largely different amounts of space without losing their specific and distinctive properties. It may be added, that there is a tendency among scientific men to believe that the bodies which are now generally regarded as elementary are not really elementary; and to look upon hydrogen, or some other body not yet discovered, as the universal element from which all the bodies now termed elementary, and necessarily therefore all compound bodies, are derived by the aggregation of the elementary molecules into groups which are special for each such derived body.

It is impossible to suppose that such aggregates of molecules could come into existence if merely inert matter were concerned. It is in fact essential to the completion of the atomic theory to assume the existence, in relation with these ultimate molecules, of some force or forces which cause their aggregation, and which directly or indirectly endow them with specific properties.

But what are these ultimate molecules? Are they really solid particles, indivisible, unalterable, and structureless? Or are they, as some have taught, mere points of concentrated force; or, as is now largely held, the rotatory portions of, or vortices in, a universally diffused something? In reply I can only say that there is, so far as I can see, no absolute proof that the atomic theory is true. It is merely a provisional hypothesis—doubtless the simplest if not the only one at present conceivable—which links together consistently, and affords a reasonably intelligible explanation of, the facts of chemistry and physics as these are at present understood.

Force, equally as matter, we know only by its properties. Many different kinds or phases of force are recognised; such as the force of gravity, which draws all relatively distant objects towards one another; cohesive force, which gives solidity to solid bodies; chemical force, to which the union of simple or multiple atoms of different elements or different substances is due; heat, light, electricity, magnetism, vital force, and so on. But some of these varieties of force have been shewn to be correlative or mutually convertible; and, indeed, it seems just as probable, as that all material bodies are derived from one elementary substance, that all forces, however different or even opposed to one another they may seem to be, are only different manifestations or conditions of one simple all-pervading power. We know that force implies movement or change; and it is even assumed, on apparently sufficient mathematical proof, that the visible motion which the application of force imparts to visible bodies is the correlative of more or less violent molecular movement in the atoms of which such bodies are composed; which molecular movement, when sufficiently violent, causes heat and light and chemical change. If such views be correct, movement, molecular or massive (or to use the technical word "molar"), is the physical evidence and measure of force.

But still the question "What is force, and what is its relation to matter?" obtrudes itself. It is generally held that force exists only in relation with matter, that it is in fact a property or function of matter and therefore cannot exist in empty space. If, however, we accept the view that the ultimate molecules of matter are mere centres or points of accumulated force, it would seem to follow as a corollary that force in an attenuated form may pervade all space. But, assuming the literal truth of the atomic theory, and that force exists only in relation with matter, what is the nature of this relation? How is it that massive bodies at a distance from one another are mutually attractive? In what sense is it that electric force or the electric current shoots in the form of lightning from cloud to cloud, or between cloud and earth, or flashes along the telegraph wire? Must we assume that, in the first case, massive bodies are surrounded, so to speak, by an atmosphere of force; and, to descend from big to little, that all ultimate atoms are enveloped in like manner; and that it is through the instrumentality of such atmospheres that simple atoms blend into aggregates? Must we assume that, in the latter case, force is transmitted from particle to particle of the air or of the wire, as the case may be, either as an independent stream or in the sense that the atmospheres of force connected with the implicated particles are passed on from one to the other and so collectively constitute a stream of force; or otherwise that the force developed or awakened in any one particle leads by a kind of catalytic action to a similar development or awakening of force in its immediate neighbours and so on, until something occurs to arrest or modify the progress of events? In other words, is force inherent in, and inseparable from, solid atoms in the sense that each atom is endowed with its own specific quantity of force, existing either actively or potentially, and which is incapable of increase or decrease, and is as permanent and indestructible as the atoms themselves? Or is it a something which, though tending to accumulate in or around such atoms, is capable also of passing through space, and thus from one atom or group of atoms to others, and consequently therefore of existing in otherwise empty space? It may be urged, and I am not prepared to dispute it, that these are all idle speculations; that in speaking thus of force I am suggesting that force is material, or at any rate subservient to the laws that govern matter; whereas of necessity force is an immaterial influence of which we know nothing and are incapable of knowing anything save through its effects on matter. But, on the other hand, we have no knowledge of matter excepting through force.

The question, nevertheless, seems to have a very profound relation to the further question as to what is life, what is living matter, and what are their mutual relations.

So far as we know living matter, or protoplasm, is composed of, and may be resolved into, the selfsame elements as contribute to the constitution of the unliving matter which forms the great bulk of the ponderable universe, and has been developed out of them. Further, as chemists show, living matter is composed of only a few out of the many recognised elementary bodies, and consists mainly of only four, the molecules of which, however, appear to be combined or associated with a degree of complexity to which no chemical combinations in the inorganic world show the slightest approach. But living matter, even in its simplest forms, manifests other characters besides these. It is endowed with life, that is with force or forces which appear to be inherent in it, and in virtue of which it can change its form and station, it can assimilate the inorganic or other matters which surround it, and transmute them into its own tissue, it can grow, it can develop, it can multiply. And moreover the progeny, to which it gives rise by gemmation or fission, present, under similar external conditions, the self-same structure and the self-same vital properties as does the parent being.

Now it is obvious that we have here a marvellous compound, so to speak, of matter and of force: of matter which, though derived from, and reducible to, simple inorganic elements, is so artfully arranged as to be something entirely *sui generis;* of force or forces which, although they may be the same as those which habitually influence inorganic matter, seem to be of a different kind, or at any rate act in a manner which seems to have no parallel in inorganic nature. Vital force (to use a convenient term) co-operating with suitable matter, under gradually developed suitable conditions, would seem: first, to coerce or guide such matter into complex combinations, otherwise organic tissue; and then, so to act upon this tissue as to produce in it all the manifestations of life which have just been enumerated, including the marvellous powers of carrying on its own life and of giving origin to other organisms, themselves formed on the pattern, and perpetuating the special qualities of, the parent. It may be assumed that neither force nor matter is absolute master of the situation, that, while matter is being guided and moulded by force, force is limited and influenced in its action by the inertia which matter opposes.

But life implies more even than has yet been hinted at; it implies also the constant wearing away or destruction of the living tissues, and the constant replacement of the resulting waste or effete products by other matter which is obtained from without, and becomes incorporated into the living mass, and consequently itself endowed with life. Thus living bodies are undergoing constant change, so that sooner or later possibly, even though still retaining their individuality, nothing may be left them of their original constituents. Indeed, every living thing has, in virtue of the life within it, only a limited duration; in other words, molecular and individual death are the natural and necessary correlatives of individual life. But if this be so, and each particle of matter is endowed with its own specific force which it cannot yield up or be deprived of, it follows: that the collective forces which constitute life must themselves change as matter changes; and that, exactly as the living body at one time consists in greater or less proportion of different matter from that which composes it at another time, and yet retains its specific identity, so the force or forces which constitute its life undergo proportionate variations, and those which give it life at one time are not precisely identical with those which give it life at another time. If it be not so we must admit, either that force exists independently of matter, and may be transferred from molecule to molecule, or from mass to mass, or that life or vital force is a something *sui generis* which pervades nature, and whose only relation to matter is that it coerces it into special combinations which are suitable for the display of its own special attributes.

I have hitherto been thinking only of such simple organic matter and such simple life as are presented by the amœba. But my argument seems to me to apply equally to all things endowed with life, even the most complicated and the highest. It is, indeed, more difficult to bridge over the gulf which separates inorganic matter from the lowliest living organisms than it is to pass step by step from these to the highest. As we pursue our investigations we find that, no matter to what height in the scale animal or vegetable life attains, life still remains associated with the same, or apparently the same, kind of elementary living matter; but we find, additionally, greatly increased complexity of arrangement and structure, due to the fact that the higher and more capable living things become, the greater is the need for differentiation of structure to subserve specific functions, which in the lower and simpler organisms are performed collectively, but less elaborately and in a more rudimentary manner, by simple homogeneous protoplasm. Vital force would seem to be the same in quality in all cases, but guided as to its action in different directions and for different objects according to the structure, the duties, and the relations of the different component parts of the organism.

A difficulty no doubt suggests itself when we come to apply the above arguments in explanation of the phenomena connected with the nervous system of animals—a difficulty arising out of the elaborate and mysterious character of the mechanism of this system and the subtle and dominant character of its functions. Yet there is no sufficient reason, so far as I can see, why, if the views just enunciated be admitted in explanation of all vital phenomena excepting those connected with the nervous system, they should not be equally applicable to the relations between nervous organs and nervous force, so far as these are merely subservient to the conscious mind. For we find here, as in other systems of organs, vital tissues built up of protoplasm, which are developed by slow degrees, and which, under the influence of suitably applied force, discharge duties which, if more specialised and far-reaching than the duties performed by other organs, are still only such duties as the nervous apparatus is, by its elaborate and special structural arrangements, fitted and adjusted to perform. Or, to express myself in other words, I can see no just reason why so much of the nervous system in the higher animals, as comprises the medulla oblongata, spinal cord, and peripheral nerves, and so much of the nervous functions as these discharge, should be regarded in a different light (so far as concerns the relations between matter and force) from the digestive organs and their functions, or the heart or lungs and their functions.

The main difficulty in the way of the acceptance of the hypothesis, which I am endeavouring to formulate, as a general and sufficient explanation of the relations between life and living matter lies no doubt in the existence in relation with the cerebrum of a conscious mind, as distinct from mere unconscious nervous automatism.

That the cerebrum or some part of it is the organ of the mind is universally admitted, and is supported by numerous considerations : among the more important of these being the facts, that it exists only in the animal kingdom ; that it increases in bulk and in complexity as animals rise in the scale of intelligence, until it culminates in both these respects in man ; that diseases affecting the brain are specially characterised by more or less serious impairment of the mental functions ; and, perhaps most important of all, that anatomico-physiological investigations have proved that the grey matter of the cerebral surface admits to a large extent of being mapped out into areæ which have severally definite relations to specific sensory or motor functions.

It is interesting to observe in this connection, that the brain, undergoing gradual development during fœtal life, has at the time of birth attained its full anatomical development, and is potentially as well adapted for the discharge of its special functions as is the adult brain, and yet that down to that epoch no evidence whatever has been shown of the existence of a conscious mind. After birth, however, a sudden and momentous change takes place. The organs of sense, the inlets to the mind, become at once exposed to those external influences which are severally adapted to act upon them; and, through their agency, impressions more or less vivid, and in rapid sequence, are conveyed to the cerebral cortex, and thus impart to the mind a knowledge of the body which it animates and of the circumambient facts of nature. At the same time also the mind, through the brain, begins to exercise its voluntary powers over muscular movements: at first fitfully and vaguely but gradually with increasing intelligence and purpose, and thus, further becomes cognisant of the autocratic power it possesses over the motor mechanism of the body, and its relations to things which are external to it. By such means knowledge accumulates, and voluntary muscular co-ordinations become more and more elaborate and exact, and the education of the mind advances.

But the above epitome by no means comprises all the conditions which regulate mental growth. For, first, the crude impressions derived from the senses are liable to be modified or idealised; second, the impressions conveyed to the brain are made, so to speak, in bulk, and are not, as a rule, analysed by the mind as the scientific student learns to analyse them, and similarly the movements which the mind directs are usually not movements of single muscles, but groups of movements co-ordinated for special purposes; third, the mind has the power of comparing and analysing and putting into new combinations the impressions which reach it from without, and thus of creating, so to speak, new sensory impressions, which are and remain as much the property of the mind as are those derived directly from without; fourth, although the mind at birth is an absolute blank, every mind possesses by inheritance aptitudes or tendencies which distinguish it, in a greater or less degree, from every other mind, and exert a more or less powerful influence over its development and quality; and lastly, it may be observed, that not only much of our early muscular co-ordinations, but much also of our later co-ordinations, are instinctive in the sense that, although such movements they may be checked or guided by the will, they do not originate in any effort of the will, may be carried on even when the brain has been removed or its functions are in abeyance, and appear to be due to the independent action of subordinate nervous centres.

Now, what is the relation of the conscious mind to the groups of impressions which find their way to the brain, to the groups of combined movement which emanate from the brain, and to the several other mental or cerebral phenomena which have just been enumerated? We know, as I have already pointed out, that much of the surface of the brain may be mapped out into areæ which have severally definite relations with particular sensory nerves or with special motor co-ordinations. We cannot doubt that it is in the sensory areæ that the impressions conveyed by the sensory nerves are taken cognisance of by the mind, and that it is through the motor areæ that the mind directs the performance of voluntary movements. And it follows, almost of necessity, that all our items of knowledge, whether sensory or motor, and whether derived directly or remotely from nervous impressions, are printed in some mysterious fashion on the specialised protoplasmic tissue of the cerebral cortex; that the pictures thus made are more or less permanent in character; and that if we had instruments of observation of sufficient delicacy we should be able to recognise some corresponding change in the cells of the dead brain. In other words, it would seem reasonably certain, that all the items of knowledge on which the mind feeds, and reasons, and acts, are duly pigeon-holed in the cerebral substance, that they are represented therein individually by some molecular change of a very subtle but very real character; and that in the same sense as the individual brain-cells are, or tend to be, permanent organs, so the phenomena engraven on them are, or tend to be, permanent.

But mind is force or a quality of force, and immaterial. How then does it take cognisance of the material impressions made on the brain tissue, and stored up in it? The difficulty to which I have called attention again confronts us. Is force so related to matter that every particle of matter is endowed with its special share? Is force an influence which is capable of passing from one particle to another, and so of accumulating in some particles at the expense of others? Can force exist apart from matter and independently of it? Are all forces fundamentally the same; and, if so, does the one fundamental force manifest different qualities determined by differences of molecular arrangement?

I have argued, that it is by the co-operation of force and matter under suitable conditions that things endowed with life have become evolved out of inorganic matter; that by such co-operation organic matter has undergone development from the condition of simple structureless protoplasm to beings of complex organisation, including man, and force itself has acquired in relation therewith other and higher qualities than apparently attached to it in earlier phases of development. In everything until we arrive at consciousness, and even as it seems to me is the development of the subordinate portions of the nervous system, and of the forces which actuate them, we see evidence of an orderly and progressive development or evolution. Nowhere in the whole history of life does there seem to have been any more striking departure, any more striking suggestion of the interposition of some new and special force, than was displayed in the primary development of inorganic matter into living protoplasm. The highly organised vegetable is scarcely less marvellous in its structure and endowments than is the highly organised animal; the living protoplasm in either case (excepting in its potentialities) presents little, if any, difference; in both cases, under the guidance of vital force, and through the union of the sexes, seeds are produced which, under the continued guidance of the same force, grow into living things, which perpetuate in and through themselves the patterns of their parents; and in both, under the influence of similar guidance, complex structures are developed which co-operate for the general welfare of the individual, and are held in due subordination one to another, and to the needs of the individual. It is true, of course, that animals and vegetables started, so to speak, on different careers: that animals took a more independent course; that in process of time their organisation and endowments became more and more differentiated from those of vegetables; and that especially a nervous system became superadded. But, unless we admit that for every stage of advancement some new kind of force was called into requisition, or was created, we have surely no right to suppose that any new kind of force was called into requisition or was created in relation to the nervous system. I repeat that, in my judgment, nervous functions, in so far as they are administered by the subordinate nervous centres, are the mere consequences, and the proof, of gradual evolution and progress towards perfection in the associated development of organic matter and organic force; and that we have, in the nervous system, simply a very perfect mechanism for the use of force and for its exercise in a specific and very important manner. But if this be so, does it not necessarily follow, that the same arguments apply equally to the structure and endowments of the supreme nervous centres, and that consciousness, with all that belongs to it, is an inherent and fundamental property of force, acting under certain specialised conditions?

Assuming that all different forms of force with which we are acquainted are mere modifications of some one simple elementary force, determined by the conditions under which it acts, and that consequently all varieties and phases of force are potentially present in elementary force; and that it is this same force which, under special conditions, constitutes life in the sense of building up, and imparting vital properties to protoplasm, and in the sense of determining, under analagous or similar conditions, the growth, development, and perpetuation of living things, and the nervous phenomena which, in the absence of consciousness, impel animals to the performance of quasi-voluntary movements and the discharge of quasi-voluntary functions; why may we not equally assume that consciousness, will, reason, are potential properties of simple force, which remain in abeyance so long as conditions are unsuitable or adverse, but manifest themselves with greater or less distinctness when conditions are favourable? It is clear that consciousness may be latent even when the material conditions, under which alone so far as we know consciousness can exist, are present. It is thus in profound sleep, it is thus also in fœtal life. In the former case it would seem that from some all-pervading but transitory interruption to the cerebral functions the accumulated cerebral impressions become unavailable, and the mind consequently a blank. In the latter case consciousness is absent simply because no cerebral, sensory, motor or other influences have yet arisen to evoke and feed the latent mental power.

It may, I think, be reasonably assumed from these considerations, and generally from the previous discussion :—first, that before potential consciousness can wake to actual consciousness, there must be in intimate relation with it something concerning which it can be conscious, for consciousness without knowledge of anything, with no experience, with no capability of action, with nothing to be conscious about, is a mere abstraction and practically non-existent; second, that consciousness can only arise (so far as our present knowledge enables us to form an opinion) when the facts of nature are by suitable mechanisms impressed in such a form on living matter suitably constituted and suitably arranged to allow of force reading (so to speak) the items of knowledge thus impressed and accumulated, or (in other words) that consciousness (although possibly it may exist potentially in all force and even throughout all nature) cannot exist as consciousness apart from the presence of a material brain or some other equivalent organisation.

The conclusions with respect to brain and mind which I venture to deduce from the foregoing considerations are briefly the following: First, that the brain is an organ or machine specially fitted to receive impressions made through the sensory nerves, to discharge influences capable of evoking combined movements for special objects, and to register impressions made on it and duties demanded of it; second, that consciousness is an inherent quality of force, latent under ordinary circumstances, and becoming manifest only when force comes into relation with matter suitably constituted and prepared ; third, that all mental phenomena depend on the reactions between brain and force suitably directed—force supplying the consciousness and all other active mental powers, brain furnishing the office and the plant for the receipt and transmission of telegraphic messages, and for their registration; and, lastly, that mental individuality or personal identity depends, not on permanence in the constituent element of the brain, not on unchangeableness in the vital force which actuates it, but upon the persistency and association of the impressions or pictures engraven on the cerebral substance.

The reasons for the last conclusion demand probably some further explanation than has yet appeared. No doubt we retain our personal identities from the beginning to the end of life. Yet we know that our brains as well as the rest of our bodies (and especially their vital parts) are undergoing constant molecular changes, so that at the end of a long life the original materials have probably been entirely replaced by others several times over; and we have, as I have endeavoured to show, reason to believe that the forces which vitalize our bodies, and which make us conscious beings, are similarly unstable. The last inference can scarcely be disputed by anyone who accepts the hypothesis that consciousness is a latent attribute of elementary force. For it must be so if force be inherent in, and inseparable from, matter, inasmuch as every particle of the brain which becomes effete and is cast off must carry with it the modicum of force belonging to it, and every fresh particle added must bring with it its own share. And it seems equally obvious that it must be so if force be a common attribute of matter, and yet be so far independent of it that it may pass freely from particle to particle; for in that case mental force would almost certainly be fed mainly by force reaching the brain through the organs of sense, and would discharge itself mainly through the efferent nerves, and would thus be undergoing perpetual change. A further reason in favour of this view is that our mental individuality depends mainly if not solely on memory, or the survival and overlapping of impressions which have been made upon the tissue of the brain, and on the continuity of our existence; much in the same sense as a river remains the same in virtue of the channel and scenery through which it flows, and the geographical course it takes, notwithstanding that its waters are undergoing perpetual change. Now the old man is the same individual as the child from which he has developed. Yet, if the phenomena of the long intermediate period could be obliterated, all that would remain to prove persistent mental identity would be a more or less hazy recollection of particular occurrences, recollections so vague that they might equally be the mere manufactured recollections of recent dreams. Seeing, indeed, how time obliterates the facts of our former lives, and tends to obliterate old knowledge as new knowledge is added, it seems obvious that were we to go on living for ever under otherwise present conditions, we should lose over and over again our accumulated knowledge, and therewith (excepting for the continuity of our existence) our mental personal identities.

It must be admitted that there is yet another way of regarding the mind, namely, that it is (as is generally believed) a something *sui generis*, a concrete force (if I may so express it) which has either seized upon and made captive of the brain prepared for its abode, or has developed concurrently with the development of the brain, and in either case may survive as such independently of the brain or indeed of matter. But this view rests only on the fact of personal identity, which can I think be just as well explained in another way, on the desire which most of us have for personal immortality, and on religious dogma. Moreover, there is nothing to show that the minds of human beings differ, save in degree, from the minds of lower animals, which have all an equal right with ourselves to claim personal immortality. Neither does it seem to me that this view is in accordance with the teachings of analogy or of science. It is only a belief and a hope.

I acknowledge, however, that we know nothing of life beyond the grave, and that even if we cannot, on scientific grounds, hold to the belief in personal immortality, it does not follow that there may not be a future for us as part at any rate of the universal force, or soul, which there is reason to believe pervades the universe, and gives life to matter, and is (as matter is held to be) indestructible and immortal.

In conclusion, gentlemen, let me acknowledge that I am not entirely convinced by my own arguments. I have endeavoured to reason out a consistent view of a very difficult, and for all of us supremely interesting, subject; and I trust that I have been so far successful as at least to have made myself intelligible. But I trust that I have not been equally successful in revealing the poverty and imperfections of my own mind and brain; at any rate I am not such a fool as to believe that it has been reserved for me to solve the mystery of mysteries.

ON MEMORY.[1]

BY JAMES ROSS, M.D., LL.D.

THE subject for discussion to-night is "Memory," and I presume that we are supposed to enquire into its nature, its limitations from other modes of consciousness, its laws of action, its connection with cerebral activity, and its dissolution by disease. But memory is so intertwined with every other form of mental activity, with the feelings which accompany instinctive and acquired movements, with sensations, perceptions and conceptions, with reasoning, from the simplest to the most abstract, and even with the emotions and the will, that it is simply impossible to deal with it satisfactorily in the few moments at my disposal to-night. To treat this subject with even a moderate degree of adequacy would involve the writing of a complete system of psychology, and consequently in the few remarks I have to offer I will presume largely upon the knowledge of psychology, which must be more or less common to the members of this Society. The laws of the association of ideas, for example, bear a most important relation to the acquirements of memory, but as I have nothing new to add to what is already known of these laws, it will be unnecessary for me to give formal expression to them here.

My *first* proposition with regard to memory is that it is a mode of consciousness, and consequently to speak of unconscious memory is a contradiction in terms. If a psychical act or an impression on the organism is wholly unperceived it cannot afterwards be recalled as a memory.

My *second* proposition is that our primary knowledge of memory is obtained by means of introspection. This knowledge is no doubt greatly extended by objective observation of the actions of other organisms, but my guarantee that anyone except myself possesses memory is an indirect conclusion from the observation of the exercise of the faculty in my own mind. I may also say that memory is an undecomposable mode of consciousness; in other words it cannot be analysed into elements which do not presuppose the existence of that which has to be explained. Now our primary knowledge of memory being subjective, and the faculty being an elementary and primary mode of consciousness, the term memory cannot be correctly employed in the explanation of complex objective phenomena. It has been said, for example, that the cicatrix left by an old wound constitutes a memory of the original injury, or that the persistence of this cicatrix shows that the tissue which enters into its formation has a memory of reproducing its like. In this connection we have heard of such an expression as "memory in disease." It might as well be said that an acorn has a memory of growing under suitable conditions into an oak. This may be an appropriate metaphorical or poetical use of the term, but it is not science. Memory is, like every other mode of consciousness, a correlative of certain changes in the brain, and the brain, like the acorn, manifests a tendency to grow into the parental form, but it is ridiculous to suppose that a scientific explanation is afforded of those facts of development by calling them memories. Objective science is the embodiment of our efforts to reduce all the impressions received through the senses or revived in our memories to the fundamental impressions of touch and muscular effort, or to number, extension and inertia or force; in other words, objective science is the reduction of all qualitative to quantitative impressions, and it is absurd to think that one step is taken in this direction by calling the reproduction of like by like in organic growth memories.

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[1] Read at a meeting of the Neurological Society in December, 1890.

My *third* proposition is, that memory being a mode of consciousness, it must be capable of being separated either by sharp or by indefinite boundaries from other modes of consciousness. Let us now, endeavour to mark memory off from adjacent departments of consciousness.

Adopting Mr. Herbert Spencer's classification, states of consciousness may be divided, according to their degree or intensity, into vivid and faint states, the former being in their derivation originals or presented, and the latter copies or represented. It is clear that states of memory will be found amongst the faint or represented feelings, and that the vivid or presented feelings may be at once excluded from the category. But even in this division the boundary between the two states of consciousness is by no means trenchant; the one passes into the other by insensible gradations. Suppose a person born blind to have had his sight restored by operation, and suppose that the darkened chamber in which he lies after the operation is suddenly lit up by a flash of lightning, which is practically of momentary duration, the subject would experience a vivid feeling of sight. But the impression made upon the retina persists for a considerable time after the withdrawal of the stimulus, and the vivid feeling would only pass in the supposed case by insensible degrees into the faint feeling which would constitute a memory of it ; in other words, the boundary which marks off the vivid and faint feelings in consciousness is an indefinite one.

Passing now to the consideration of more complex clusters of feelings, let us first consider those which accompany our own bodily actions. It is clear that the memory of these actions will be found amongst the faint revivals of the feelings which accompanied an act that has now receded into the past. At the outset I must draw a distinction between feelings which are directly and habitually connected with the act and those which are remotely and more or less fortuitously connected with it. Let me, for example, suppose myself walking in a particular direction, that at a particular point I see a lion at large in my way, and that, somewhat naturally, I take to my heels in the opposite direction, this would constitute an experience which would implant that walk on my memory for the rest of my life. The distinct memory of this walk would, however, be due to the fortuitous circumstance of meeting the lion and not to the tactual and muscular sensations which accompanied the use of the limbs. And similarly if I walk on slippery ground and am in danger of falling at each step, the clear recollection I have afterwards of this walk arises, not from the direct and habitual feelings associated with the act, but from its indirect or fortuitous accompaniments. In our subsequent remarks it will be understood, unless otherwise stated, that in speaking of the feelings associated with bodily actions we speak only of the direct or necessary feelings. For an example of bodily action I will take a complex act, but one which is performed under the simplest possible conditions. I allude to the act of sucking in new-born infants. When the nipple is placed in the infant's mouth it at once begins to suck. The feelings necessarily connected with the act are certain tactual and muscular sensations, and those which are fortuitously connected with it are the feelings of warmth received from contact with the mother, the feelings of taste caused by the flow of milk over the tongue and palate, and the feelings of comfort caused by repletion. That the latter feelings are fortuitously combined with the act is shown by the fact that the infant will suck a finger placed in the mouth. Now it is clear that in the first act of sucking the co-ordinated motions are not guided by a memory of past feelings, but with the second act it is possible for the infant not only to have the vivid sensations which accompany the act itself, but a faint revival of the feelings which accompanied the first act, and the fact that the child improves, within certain narrow limits, in the power of sucking shows that the act may be accompanied by a rudimentary memory. Inasmuch, however, as the act of sucking soon reaches its limits of precision, it may be presumed that the revival of past feelings in consciousness has had very little to do in perfecting it and nothing at all in initiating it. This conclusion is much strengthened by the fact that the act of sucking is accomplished by acephalous children, and, therefore, in the absence of conscious feeling. The fortuitous feelings which accompany sucking, being generally of a pleasurable kind, excite a desire for a continuance and repetition of the act, and the child may doubtless be supposed to have a memory of these feelings, but they cannot have taken an active share in perfecting the act when regarded as a co-ordination of complex movements. It may, therefore, be inferred that instinctive actions are not guided by memory, or that the memory is a rudimentary one.

In the acquisition of new actions of any degree of complexity, however, memory plays an important part. In acquiring the accomplishment of dancing a quadrille, for example, there is at first a considerable strain upon the memory. Each part of the dance is mentally rehearsed before the individual movements are begun, but by constant repetition each movement is followed by its appropriate successor without the aid of memory until at last the evolutions of the dance follow one another with automatic coherence. It is the same with all other psychical actions which are not, more or less, completely organised at birth. In acquiring the power of walking the child has, doubtless, to remember at first how to keep his balance by inclining the body at each step towards the side of the foot planted on the ground, and even how progression depends upon the alternate advance of each leg, but in a short time the separate movements cohere so mechanically that the one follows the other without any conscious effort, and, therefore, without memory. It is the same in acquiring the power of articulate speech. Each letter demands for its articulation the concurrence of a definite combination of movements of the chest, larynx, tongue, soft palate, and lips, and it is clear, therefore, that the movements requisite for effecting spoken speech must be marvellously complicated. I saw a young gentleman lately who, after an injury to the head, had become the subject of a complete motor aphasia, and, although understanding every word addressed to him, he was incapable of articulating a single letter. I requested him to say the letter "p," but he shook his head mournfully, and made no attempt to obey the request. I then directed his attention to my mouth, and in closing my lips firmly he immediately imitated the action. I now exploded my lips apart by uttering "p." He at once recognised my intention and laughed. After a few efforts he followed closure of his lips by contracting the muscles of his chest, but the blast only gave rise to a kind of grunt in the larynx, and escaped through the nose. My visit had now to terminate, and I laughingly said to his attendants, "He will soon be able to say 'p,' and then you will teach him to say 'pig.'" I was afterwards told that on the following day he voluntarily continued the exercise, but for a long time with uniform failure. At last, however, he burst out with "pig" at the highest pitch of his voice, and then laughed with the utmost pleasure at his performance. From that time he went on to acquire other sounds, but I have not recently heard of his progress. Here, then, we have the articulation of such a comparatively easy letter as "p" requiring a distinct effort of memory from a person who had a full and ample organisation of speech on the ingoing or perceptive side, and the illustration is well calculated to impress upon our minds the efforts of memory which must have been made by the child before speech had become acquired, although in the adult the words follow one another so quickly and coherently that hardly any effort of memory is required for the completion of whole sentences. In reference to this subject Mr. Spencer says :—"But the clearest instance of the gradual lapse of memory into automatic coherence is yielded by the musician. Originally, he was taught that each mark on the paper has a certain name, and implies that a particular key on the piano is to be struck, and during his first lessons each recurrence of this mark was accompanied with a distinct process of recollecting which key on the piano he must strike. By long-continued practice, however, the series of psychical changes that occur between seeing this mark and striking this key have been reduced into one almost automatic change. The visual perception of the crotchet or quaver; the perception of its position on the lines of the stave, and its relation to the beginning of the bar; the consciousness of the place on the piano where the answering key lies; the consciousness of the muscular adjustments required to bring the arm, hand, and fingers into the attitudes requisite for touching that key; the consciousness of the muscular impulse which will give a blow of the due strength, and of the time during which the muscles must be kept contracted to produce the right length of note, all these mental states, which were at first so many separate recollections, ultimately constitute a succession so rapid that the whole of them pass in an instant; as fast as they cease to be distinct states of mind—as fast as they cease to fill appreciable places in consciousness—so fast do they become automatic. The two things are two sides of the same thing. And thus it happens that the practised pianist can play while conversing with those around him, while his memory is occupied with quite other ideas than the meanings of the signs before him."

Let us now direct our attention to the consideration of the impressions made upon our senses by external agents. As I direct my eyes, for example, towards the table before me I see a patch of colour which differs from the colour of the surrounding surface. I direct my eyes rapidly from point to point of this patch and experience sensations of muscular sense connected with the ocular movements which give me information respecting its form. But besides the feelings thus presented I instantly judge that were I to walk forward a few paces and stretch out my hand this patch of colour would be found associated with properties of resistance to my movements which I call weight and solidity, and and that what appears to me as an oblong patch of colour has a certain individuality which makes it numerically different from surrounding objects. When, therefore, from the dynamical property experienced as colour the presence of the statical properties of weight and solidity is inferred, are these represented feelings to be regarded as memories? If so, by far the greater number of the attributes which rise into consciousness in every act of perception is a memory. But the rule which we found to be applicable to the memory of movements applies here also; if the cohesions between the presented and represented feelings are indissoluble so that the one follows the other in an automatic manner, then the represented feelings are not memories. The connections between the two sets of feelings may have begun as a memory in the child, but now that they have become automatically coherent they cease to be memories. That the patch of colour I see before me implies an object having weight and extension I *know*, but it would be regarded as a misuse of language were I to say that I *remember* it. And when the object before me revives in my mind clusters of previously experienced feelings, which I identify as like to the clusters of feelings now experienced—when, to use the language of Mr. Romanes I form a *generic* idea or *recept*, the process does not involve memory if the object has become so familiar that the revived experiences cohere with the present experiences, and with one another in an instantaneous manner. It is, even, possible to pass to a still further complication of mental combinations without memory being called upon to take an active share in the process. When, for example, I give to the object before me the name of book I form a concept by means of which I identify or classify the object with all the books I have ever seen or heard of, and even with the books which have not yet existed but may be expected in the future. Now the formation of concepts, and of classification in general may involve the highest exercise of memory, but when the properties of the objects classified are so familiarly known that the name when spoken or written calls up into consciousness the properties common to the whole group instantaneously then memory can hardly be said to take a share in the mental process. And similarly with regard to common and familiar sequences. The manuscript I hold in my hand I classify with heavy bodies and know that if left unsupported it would fall to the ground, but surely no one would expect me to say that I hold the manuscript in my hand because I remember that it would fall if unsupported. And although classification may, as we have said, involve the exercise of memory in high degree its tendency is by grouping like qualities to like in the midst of innumerable unlikenesses, to facilitate the action of memory. Things difficult to remember become, under a good classification, easy to remember, and things easy to remember assume by further experience automatic coherence in which memory is dispensed with. And similarly with regard to trains of reasoning, their tendency being to facilitate the use of memory and finally to dispense with it almost if not altogether. Suppose a person who had at one time been fairly familiar with the demonstrations of geometry is asked to prove the fifth proposition of the first book of Euclid, and suppose that he has not read the actual demonstration for the last twenty years. The data are two equal sides in a triangle and it is required to prove that the angles at the base are equal, and on the equal sides being produced beyond the base, that the angles on the other side of the base are also equal. The axioms of equality and the definitions required in the proof are so familiar to every one in the acts of daily life that it would require no effort of memory to recall them into consciousness, and if the person only remembered that the required proof rests upon the proof of the equality in all respect of two triangles which have two sides and a continued single equal and that Euclid proves this theorem by the ideal superposition of the triangles, the demonstration of the fifth proposition would present no difficulties. From the data the person would pass by easy steps to the required conclusion because each successive inference would only involve the simple relation of equality, and thus the demonstration would require little or no effort of memory. No one, indeed, would call such a demonstration, even under the supposed circumstances of not having read the proposition for twenty years, a high exercise of the memory.

Some people, strange as it may seem, appear to be incapable of grasping the reasonings of geometry. Suppose a person of this class had committed verbally to memory the proof of the fifth proposition, without understanding it as a concatenated system of proof, and that after twenty years he were able to repeat it correctly, I think the exercise would be deemed an extraordinary manifestation of a tenacious memory. Wherein lies the difference between these two persons? The first by recalling to consciousness a few easily remembered, because well organised, fundamental principles, is enabled to pass from the ideas of the data to contiguous ideas cohering to them by the simple relation of equality, and from them to the next contiguous ideas, and so on by easy gradations to the conclusion. But the second, in reproducing the successive ideas, has to depend upon the sounds of the articulated words, sounds which in the supposed case have little tendency to suggest one another, and upon the visual representations of the forms and positions of the words and letters on the printed page, and to reproduce them correctly in the absence of any principle of association would be justly regarded as an extraordinary exercise of memory. It may, therefore, be concluded that the more the ideas cohere in a well established system of ratiocination the less room is there for the exercise of memory. The scientific man who forms indissoluble cohesions amongst his facts and ideas by means of the fundamental generalisations of orderly classification, and the invariable relationships of systems of ratiocination, are admitted to exercise the understanding chiefly, and the memory but little, while the poet and artist, whose facts and ideas cohere in ways which are fluent and easily dissociable, and in relationships which are variable and transitory, are acknowledged to exercise the memory with its allied capacities of phantasy and imagination chiefly, and the understanding in a subordinate degree. As examples of the two orders of mind I may mention Darwin and Macaulay. Perhaps no one ever lived who had his mind stored with a larger number of diversified facts and ideas than Darwin, and yet he is never quoted as an example of a man possessed of an extraordinary memory, although no one would deny that his memory was active and tenacious. Darwin approached the phenomena of the universe from the stand point of a naturalist, and his facts were held together by means of the fundamental generalisations of morphology. Even in recalling to his mind the special adaptations of individual organisms, and of rarely occurring varieties, he was aided by the fertile principle of the survival of the fittest so that he could mentally pass from one modification of structure to another by easy gradations, and it is, therefore, recognised that in him the memory was subordinate to the understanding. The mind of Macaulay was possibly less amply stored with facts and ideas than that of Darwin, and yet his feats of memory have never ceased to be the wonder of succeeding generations. Inasmuch as Macaulay did not direct his attention to the great generalisations of sociology which underlie development in history, but contented himself with the province of a brilliant descriptive historian, it will be admitted that the facts and ideas which occupied his attention were held together by cohesive bonds much more easily sundered than those which occupied the thoughts of Mr. Darwin, and consequently in storing the facts in his mind a great strain would be made upon his memory. And, indeed, the examples usually given to show the extraordinary tenacity of his memory represent him as recollecting a large number of facts in the entire absence of any natural bond of union or native coherence. Trevelyan says of him: "He was always willing to accept a friendly challenge to a feat of memory. One day, in the Board Room of the British Museum, Sir David Dundas saw him hand to Lord Aberdeen a sheet of foolscap, covered with writing arranged in three parallel columns down each of the four pages. This document, of which the ink was still wet, proved to be a full list of the senior wranglers at Cambridge, with their dates and colleges for the hundred years during which the names of senior wranglers had been recorded in the University calendar. On another occasion Sir David asked: 'Macaulay, do you know your Popes?' 'No,' was the answer, 'I always get wrong among the Innocents.' 'But can you say your Archbishops of Canterbury?' 'Any fool,' says Macaulay, 'could say his Archbishops of Canterbury backwards,' and he went off at a score, drawing breath only once in order to remark on the oddity of there having been both an Archbishop Sancroft and an Archbishop Bancroft, until Sir David stopped him at Cranmer."

The very essence of such an exercise, regarded as a feat of memory, lies in the circumstance that each name stood for a group of facts which were almost entirely isolated from the group of facts embodied in the next name. In other words, the first name in the series had, neither by sound nor by meaning, any natural tendency to suggest the second, nor the second the third and so on. The ideas represented by the names were not bound together by any natural bond of cohesion. In Macaulay then the memory and the imagination appeared to predominate over the understanding, although no one would deny to him excellent reasoning faculties also.

I find from Sir W. Hamilton that the distinctions which I have endeavoured to draw between memories and the thoughts which rise into consciousness in an automatic manner was recognised by Aristotle. "Reminiscence," he says, "takes place in virtue of that constitution of the mind whereby each mental modification is determined to arise as the sequel of a certain other." He drew a sharp distinction between sequences which are necessary and those which are habitual only. As an example of necessary reminiscence he adduced the fact that he who is reminiscent of fire is at the same time reminiscent of heat, and his learned expositor thinks that by necessary sequences he meant such ideas as cause and effect, means and end, premises and conclusion, these being relations the one of which involves the other. In his subsequent treatment of reminiscences Aristotle drops the necessary sequences, and treats only of the habitual. He divides these into (1) intentional or voluntary, and (2) unintentional or spontaneous reminiscence. By *unintentional* reminiscence he means the order in which thoughts of past events arise in the mind in ordinary thinking, and by *intentional* the order in which the ideas arise when we voluntarily seek out a remembrance. But as the laws which govern intentional and unintentional reminiscences are the same this distribution may be neglected. Now let us attend to what takes place in an ordinary act of recollection. Talking to a friend lately of a mutual friend I said: "I saw him last in the autumn of 1888," the date being added after a slight hesitation. Before I was able to fix the date accurately my mind passed through a rapid succession of reminiscences. I remembered that I met my friend while travelling from Glasgow to Aberdeen. My visit to Glasgow was on the occasion of the meeting of the British Medical Association in that town. The Glasgow Exhibition was then open. That Exhibition was held in the year following the Manchester Exhibition. The Manchester Exhibition was held in the Jubilee Year. The Queen commenced her reign in 1337, and to this date add 50 and 1 year, and it brings us to 1888 when I met my friend. Now, circuitous as this route to the reminiscence is it was much more circuitous in reality, inasmuch as each successive step in the act was held together by minor reminiscences which passed so rapidly through the mind that it would be difficult to reproduce them. The date of the commencement of the Queen's reign, for example, is automatically impressed upon my mind by a certain relation it bears to the date of my own nativity.

The above example of a reminiscence leads me to speak of the relationship between memory and emotion. So far we have only spoken of frequent recurrence in experience as the chief factor in facilitating the act of memory, but when an isolated event engages the emotions its occurrence may be as deeply engraven on the memory as if it had been frequently repeated. I shall never forget once seeing a man drowned from the painful feelings it aroused. And similarly with regard to the determinations of the will. I shall never forget certain critical periods in my own history in which it appeared to me that great issues depended upon whether I should determine to act in this way or in that. And when we examine into the memory of our individual lives does it not consist chiefly in the revival in consciousness of a succession of ideas and images of events and scenes in which our emotions were powerfully engaged? The recollection of our own individual history is made up memories of such events as the first joyous experiences of childhood, the commencement of school life, the going to college, the obtaining a degree, the entrance upon practice, the beginning of married life and the birth of our children, while the picture is filled in by remembrances of the painful feelings aroused by parting with friends, and by the disappointments of life generally, and of the joyful feelings aroused by friendly greetings, and by personal conquest, acquisition, and success. Our remembrance of the history of our country is in no way different. The events which are calculated to strike the imagination, the great and decisive battles, the careers of prominent statesmen and rulers, the great State trials, and the important legislative enactments, are revived in memory with facility, and the minor details cluster around these. It will thus be seen that, on the one hand, the co-existing and successive events which cohere in our ordinary acts of recollection are highly complex, are now past, are not likely to recur, and are held together in fortuitous combinations; while, on the other hand, the events which have ceased to be part of memory are those which have recurred so frequently in experience that they are held together in necessary and invariable bonds, or in bonds which are not readily dissociable. I shall now endeavour to interpret the above analysis of memory in terms of molecular changes in the substance of the brain.

A remembrance implies, as we have seen, consciousness, and to be conscious of feelings, or of relations between feelings implies that the feelings themselves have occupied a perceptible duration. The psychical states, which have no appreciable persistence, are not attended by consciousness, and *a fortiori* do not enter into memory. Memory thus belongs to that class of psychical states which are in process of being organised. It is only when a resistance is offered to the flow of energy from the impressions made on the surface to the muscular excitations necessary to effect the appropriate bodily adjustments, or from the presented impressions to the nervous changes which are the co-relatives of the represented impressions, that the psychical state is a conscious one, and can afterwards become a part of memory. When the nervous connections become so fully organised that the resistance to the flow of energy ceases the psychical states have no appreciable duration, and cannot be regarded as a part of memory. The more complicated the impressions are the more resistance is offered, other things being equal, to the onward flow of energy from the senoury inlets to the motor outlets, and the more are the corresponding psychical states attended by consciousness. Many of these complicated impressions, not being repeated again in experience, fade from the memory; but those of them which are so profound in strength that they are attended by a strong emotional disturbance, become permanently stamped upon the organisation, and subsequently recur in the mind as memories. And when one such complex experience calls up other experiences which are also complex and not frequently repeated, the cohesion between them are feeble, and the transitions, taking an appreciable time, the answering psychical states become part of memory.

The dissolution of the brain in disease affects the memory in various ways. Every one knows that the facts of a particular science, which have been hastily crammed for the purpose of passing an examination, quickly fade from the memory. The organisation of the facts has been incomplete, and all traces of them soon disappear. In old age, when the nutrition of the brain begins to fail, the memory for recent events becomes imperfect, while the reminiscences of the impressions of childhood are still active. After concussion of the brain all the events which had occurred from a certain date, some weeks or even months prior to the injury up to a date considerably after it, may be entirely forgotten, while the events which had occurred anterior and posterior to these dates are remembered. After other injuries a man may utterly forget the use of one language, while retaining the full use of another. The most remarkable disorders of memory in disease are, however, to be found in the various forms of aphasia. The boy whose case I have already mentioned, who could not articulate the letter p, although he had the full use of language on the perceptive side, may be said to have forgotten the Kinæsthetic impressions which are associated with the combination of movements necessary to effect the articulation. He could not rehearse the movements in thought any more than he could accomplish them in act. In word blindness again there is a profound disorder of the visual memory. The sight of the written words do not call up the sound of the words, or the idea of the things signified. In minor degrees of this defect the subject cannot recall a visual representation of the scenes he has visited, and may be unable to form a mental picture of his wife and children, and may even forget the appearance of his own countenance. But even in such a case the automatic visual cohesions remain unimpaired. The sight of a key, for instance, immediately revives not only the idea of its solidity and weight, but also its uses. When, however, the injury is very profound the sight of an object fails to revive in consciousness its uses, while still reviving its general characteristics with regard to its statical properties of weight and resistance. In cases of general paralysis recorded by Fürstner the patient knew that a coin was a piece of metal, but failed to remember its uses as a money tender. In the dogs operated upon by Goltz the sight of a leg of mutton revived the idea of its resistance, so that it was avoided as an obstacle, but failed to recall the more complex and special idea of its use as food.

In word deafness the chief injury is done to the auditory word images. The sound of the word fails to revive in consciousness the corresponding idea. The patient can still hear noises, and recognises music, but words have ceased to have any meaning for him. It is unnecessary to multiply examples. I do not pretend for a moment that the view of memory here adopted is in any way original. I fear I cannot claim for it even originality of treatment, but I trust that it at least forms a coherent whole, that it gives an intelligible account of the growth, development, and decay of memory, and that it will form a basis for a profitable discussion of the subject.

ON SOME POINTS IN THE ACTION OF MUSCLES.

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In the examination of cases of paralysis in the upper limbs of different groups of muscles, there is often difficulty in ascertaining which particular muscles are the ones affected in any one case; during the course of this examination conditions have been brought out which are at variance with the views commonly accepted, and also some points which I believe are novel, I have therefore thought it advisable to publish the results with the hope of bringing them before the Neurological Society for the benefit of its criticism.

For this purpose I would propose the following questions:

1. Which method gives the most exact results in determining the action of any given muscle?

2. Do the antagonists of a muscle always contract when the muscle is thrown into voluntary action?

3. Does a muscle ever produce a movement which is diametrically opposed to the usual action of this muscle?

4. May a muscle, which can produce two different movements, be paralysed for the one movement and not for the other?

5. If the last question be true, what is the practical diagnostic value of this condition?

1. Taking the first question, viz., which method gives the most exact results in determining the action of any given muscle, there are three different methods used—(*a*) the dissecting-room method, *i.e.*, putting the limb in a certain position and then pulling on the muscle along its line of action and noting the change which takes place in the joint on which the muscle acts; (*b*) the electrical method, first

used by Duchenne, where the limb on the living body is put into different positions, and on stimulating the muscle by the faradic current the resulting movement is noticed; (*c*) the method (which may be called the natural one) in which definite movements are performed by the living subject, and the muscles which are thrown into action are noted.

In examining these three methods it will be seen that the first is the only one which can be used in the case of muscles so deeply placed as not to be visible when they contract, or not to be reached by the faradic current.

But both the anatomical and the electric methods are open to this serious error, that it does not follow because a muscle, when pulled upon or excited electrically, produces a certain movement, that this muscle is necessarily called into action by the will during life to perform this movement. In illustration of this I would take as examples two muscles, viz., the supinator longus of the forearm, and the upper or clavicular fibres of the pectoralis major. With regard to the supinator longus its action is differently stated by various authors. According to Duchenne,[1] who examined it electrically, it "is a powerful flexor of the elbow joint, it is not a supinator but a pronator of the forearm, but the movement of pronation is much less than that of supination by the biceps." According to Ellis[2] its chief use is to flex the elbow joint, but if the radius is either forcibly pronated or supinated it can be put midway between pronation and supination. The late Mr. John Marshall[3] described this muscle as "acting slightly as a supinator, most so when the forearm is flexed at the elbow, but it is evidently a powerful adjuvant flexor of the elbow joint."

According to Prof. MacAlister[4] the supinator longus "is a flexor of the elbow joint and a supinator when the hand is fully pronated."

It will thus be seen from the above that though all authorities agree that the supinator longus is a flexor of the elbow joint, some consider it to be a supinator, others a pronator, while according to some anatomists, like Mr. Ellis, the muscle was considered to be both a pronator and a supinator according to the position of the hand.

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[1] "Physiologie des mouvements," 1867, p. 128.

[2] "Demonstrations of Anatomy," 11th Edition, p. 313.

[3] "Anatomy for Artists," 1878, p. 367.

[4] "A Text-book of Human Anatomy," p. 295.

The discrepancy which is found to exist in the accounts of various authors can be settled by using the method which I venture to think gives the only reliable result, viz., to examine the action of this muscle on the living body when thrown into voluntary movement, and for this purpose the most exact results are obtained from an examination of one's own muscles or those of another medical man, as we can thus ensure that one movement—and only one—will be carried out.

With regard to the supinator longus, I have found when the forearm is strongly supinated or pronated against resistance, and when care is taken not to flex the elbow joint, that no contraction of this muscle can be made out, whereas it instantly contracts and becomes prominent when the slightest flexion of the elbow is made. I have, therefore, come to the conclusion that this muscle is a pure flexor of the elbow joint, and is neither a supinator nor a pronator.

Another muscle, whose action has been investigated electrically by Duchenne,[1] is the upper or clavicular fibres of the pectoralis major, who found that when "the upper arm is raised vertically and the upper part of the muscle is made to contract (electrically), the limb is seen to be carried from behind forwards, to approach the middle line, and to be depressed as far as the horizontal line"—in fact this muscle is a depression of the uplifted arm. On trying this movement on my own arm and depressing it voluntarily from the vertical position against resistance I fail to feel any contraction of the clavicular fibres, though they can be instantly brought out in contraction, when the arm is elevated from the advanced horizontal position, and I am constrained to believe that the depressor action of this muscle, obtained by electrical stimulation, is not used in voluntary movements. If this be correct, it shows that although a muscle *can* perform a certain movement it does not at all follow that it *must* be called into action on voluntary movement.

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[1] *Op. cit.*, p 91.

It therefore seems in the case of both the supinator longus and the clavicular fibres of the pectoralis major that erroneous results may be obtained if we trust only to the movements produced by electrical stimulation or by the dissecting room method, and that the only certain method of obtaining accurate results is by examining the muscles when thrown into voluntary movement in the living body.

2.—Do the antagonists of a muscle always contract when this muscle is thrown into voluntary action?

The theory that the antagonists always contract when a muscle or set of muscles are moved voluntarily—as for instance that the triceps is always put into action when the elbow is flexed by the biceps, brachialis anticus, and supinator longus—has been strongly maintained by Duchenne,[1] who has stated "that all voluntary movements of the limbs and trunk result from a double nervous excitation, in virtue of which the two orders of muscles, which in association have a contrary action, are put simultaneously into contraction." Duchenne also quotes Winslow as the first observer to describe the muscles, acting on a joint, under the three heads of motive, moderating, and directing.

From observations on myself and on others I venture to think that this statement of Duchenne's is perhaps rather too comprehensive to apply to all cases. Taking the upper limb as affording the best situation for examining this question, it is difficult to obtain a definite result with regard to the muscles moving the digits and the wrists, as it is not very easy to feel or see whether the extensors of the fingers are contracting when the flexors are in action. And here it would be well to distinguish between the actions of the extensors of the fingers and the extensors of the wrist; it is well known that to enable the flexors to act to advantage the extensors of the wrist should be able to contract so as to fix the wrist, but in so doing they are not antagonists to the flexors but they are synergic with them; the true antagonists to the flexors are the extensors of the fingers. In the delicate movements of the hand generally, the co-operation of the extensors and flexors would doubtless be required to produce great precision of direction and power.

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[1] *Loc. cit.*, p. 766.

It is, however, in the flexors and extensors of the elbow that more exact observations can be made, owing to the facility with which these muscles can be felt and seen. According as the movements are rapid and violent or slow and weak, so the manner in which they are performed vary. Taking violent movements first, if a violent attempt be made to extend the elbow against resistance it seems quite impossible either by palpation or visually to say that the biceps or supinator longus is contracting at all. At rest with the elbow flexed to a right angle, the biceps is quite flaccid and can be freely moved from side to side, and when the triceps is contracting vigorously against resistance with the elbow kept at a right angle, no change can be made out in the biceps from its condition at rest.

The same thing can be seen in the case of violent flexion of the elbow by the biceps, &c., when the triceps can be felt to be relaxed. Also in the case of the shoulder, the clavicular fibres can be felt to contract violently while their antagonists are quite flaccid. So with regard to the extensors and flexors of the knee, if the knee be extended and the extensors be relaxed so that the patella be moveable from side to side, then on violently trying to flex the knee against opposition the patella can still be moved, whereas the slightest contraction of the extensors, in beginning to lift the heel off the ground, instantly fixes the patella. In fact in cases where it is difficult to make the patient understand to relax his extensors in trying the knee jerk, I have succeeded in obtaining it by making the patient violently flex the knee against resistance, when the extensors are relaxed.

Another familiar instance of violent muscular action unrestrained by their antagonists is afforded, when an empty can is supposed to be full of water, and the amount of force is put out requisite to lift a heavy weight, and it is not till the can is lifted violently up that the action is restrained by a fresh movement.

From the above observations it seems in violent actions that when the muscles are contracted, the antagonists cannot be felt or seen to be in action.

With regard to slow moderate movements the mechanism is not quite the same, and it varies according as the movement is in the direction of the natural tendency of the limb to fall. For this purpose the following method is useful in examining the manner in which slow movements are performed by the elbow. The humerus is to be placed and kept in a horizontal position, and the elbow is to be completely flexed so that the hand touches the shoulder. If now a weight be put into the hand and the elbow be slowly extended voluntarily the triceps is felt to contract, while the biceps is not altered and feels quite relaxed. This condition remains until the forearm becomes vertical, *i.e.*, at right angles to the humerus,when the triceps ceases contracting, and the biceps takes on action and allows the elbow to be gradually extended ; in fact, it appears to prevent the hand and its weight from falling too rapidly. During this latter half of the action no contraction of the triceps can be made out. Whereas when rapid extension is made the triceps contracts throughout the movement till the elbow is completely extended. It thus appears that although in slow movements the antagonists, *i.e.*, the biceps, contracts, it does so to retain the movements produced by the weight becoming too fast, and I am not able to satisfy myself that the biceps and triceps are contracting at the same time.

3. Does a muscle ever produce a movement which is diametrically opposed to the usual action of this muscle?

In considering this point it is obvious that the above query could not be applied to a hinge joint such as the elbow, as a muscle like the triceps could not be imagined to have any other action on that joint but extension. It is, however, on joints like the radio-ulnar or the shoulder joint that this double action might take place. In the former of these, the supinator longus, as stated above, has been considered by some authorities to have the power of both supination and pronation, but from my observations I find that this muscle has not the power to perform both these movements, and further that it neither supinates nor pronates. In the case of the shoulder joint the upper fibres of the pectoralis major have been stated by Duchenne[1] to be "at the same time elevators and depressors of the arm." There is no doubt that the upper part of the pectoralis major elevates the advanced arm and carries it to the vertical position above the head, but, as already stated, I have failed to find any contraction of it when the arm is forcibly depressed from that position downwards and forwards, although it is in the position to do this, and has been found to depress the arm when stimulated by the Faradic current. According to Duchenne the upper fibres depress the uplifted arm as far as the horizontal line, and the lower fibres carry on the movement below this point, while from my observations the lower fibres alone depress the uplifted arm, and not the upper fibres at all. According to MacAlister (*op. cit.*, p. 540) both sterno-mastoids can draw the head backwards, but I have failed to find any contraction in extreme extension of the head and neck.

I have examined other muscles in the body, but I have been unable to find any instance where any muscle or group of muscles produce a movement which is diametrically opposed to their usual action.

4. May a muscle which can produce two different (not opposite) movements be paralysed for one movement and not for the other?

The above condition has been observed in the muscles of the eyeball, where the internal rectus of both eyes may be paralysed by a central lesion for the action of converging the eyeballs, and yet each muscle may be capable of acting with the opposite external rectus in producing conjugate movements in a lateral direction, but as far as I am aware this condition has never been shown to exist in the muscles of the limbs.

As the muscle in which I first observed this condition was the pectoralis major, it would be well first to say a few words about its action.

The best account is that given by Mr. John Marshall,[2] who states that it "draws the humerus across the chest, rotating it inwards; . . . conjointly with the latissimus dorsi and the teres major the middle and lower parts of the pectoral bring the arm suddenly downwards from the elevated position as in chopping, but the upper or clavicular portion distinctly helps in raising the arm and fixing it in a forward position."

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[1] *Op. cit.*, p. 92.

[2] *Loc. cit.*, p. 344.

It would thus appear from the above that the pectoralis major is really two muscles, which, when they act as a whole, adduct the humerus and carry it towards the middle line, but when each part acts by itself, its action is antagonistic to the other, the upper fibres elevating, the lower depressing the humerus. Duchenne[1] was one of the first, if not the first, to describe the pectoralis major as really consisting of two muscles, "whose functions are quite distinct, and which often contract separately for special purposes" (p. 92), and further on "the upper fibres carry the arm horizontally forwards, ... and it is the chief agent, the deltoid only being called upon to maintain the horizontal position" (p. 94). Allusion has already been made to the supposed depressor action of the superior fibres.

The inferior part is described as always a depressor, and (p. 99 *loc. cit.*) he distinctly states that he does not "believe that the inferior fibres of the pectoralis major are associated with the superior fibres in carrying the advanced arm inwards, even in powerful movements, for the latter would become in that case antagonistic to the horizontal elevation by the deltoid."

The best way to bring out the various actions of the pectoralis major on oneself, or on patients, is to voluntarily abduct the humerus away from the chest wall by the deltoid to the horizontal line, then to slowly carry the arm horizontally forwards towards the middle line, and at a certain angle, which varies in different people—in my own case the humerus makes rather more than a right angle with the front of the chest—the upper fibres of the pectoralis major suddenly start out and carry on the movement. If now the advanced humerus be voluntarily forcibly carried against an obstacle towards the middle line, the whole muscle, including upper and lower fibres, contracts vigorously. Again, if the horizontally-advanced arm be raised upwards against an obstacle, as in holding a dumb-bell out straight in front, the upper fibres alone contract, and the lower sternal fibres relaxing, the sharp edge of the contracting upper fibres can be readily felt and seen ; on the other hand, if the humerus be then depressed from that position by pressing down on a mantel-piece, the upper clavicular fibres are relaxed, and the lower sternal fibres are vigorously contracted.

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[1] *Loc. cit.*

The muscle can, therefore, perform three different movements—adduction of the humerus to the middle line when the whole muscle acts, elevation of the advanced humerus above the horizontal line by the superior clavicular fibres, and depression of the arm in any position by the lower sternal fibres.

It will now be readily imagined that one of the above movements might be paralysed without the others being affected, and it was quite by accident that I discovered this to be the case.

About two years ago I saw a patient (shown at the Neurological Society) at the Great Northern Central Hospital, who, while jumping over a stile, fell down and hurt his left shoulder, and in the country the arm was put into splints ; on taking these off it was found that the boy had paralysis of the supinator longus, biceps, brachialis anticus, and deltoid ; there was no anæsthesia, and it seemed as if it were a case of polio-myelitis, but whether it occurred at the time of the fall or subsequently could not be ascertained. When I saw him he had absolutely no power to abduct or advance the left humerus, and there was not the slightest attempt to perform these movements either in the deltoid or in the upper fibres of the pectoralis major. As the deltoid did not re-act at all to the faradic current, I expected to find that the upper fibres of the pectoralis major would also not re-act, but to my surprise this muscle re-acted normally (perhaps not quite so strongly) to the same current as on the other side, and also normally to the constant current. It was then found that when the patient was told to *adduct* the humerus to the middle line, he could do so with normal strength, the whole muscle, including the upper clavicular, as well as the lower sternal, acting well, whereas when he tried to elevate the advanced arm, the whole of the pectoralis major ceased to act.

In this case it was evident that the upper fibres of the pectoralis major could act in association with the lower fibres in adducting the humerus, but they could not be put into action to perform the movements of elevation in association with the completely paralysed deltoid.

Several examples similar to the preceding have come under my notice during the past two years, and lately a case of polio-myelitis, under Dr. Hughlings Jackson; also one of amyotrophic lateral sclerosis, with complete paralysis of the deltoids on both sides, in whom, although there was not the slightest attempt made by the upper fibres of the pectoralis major to elevate the arms, when placed straight in front of him, he could adduct the arms to the middle line with considerable power, by both parts of the pectoral.

In connection with these observations it is interesting to note that, although Duchenne states (*loc. cit.* p. 93) that the upper fibres of the pectoralis major carry the arm horizontally forwards, he says further on (p. 96) that the deltoid performs this movement by its anterior fibres. He bases this idea, which is rather in opposition to what is stated on p. 93, on the fact that he has seen cases in which the deltoid was paralysed, and who could not advance the arm to put the hand on to the opposite shoulder, but in whom the pectoralis major was not paralysed (presumably for other movements); while, on the other hand, he had a case who could cross the arms in front of him when both pectorals were paralysed, but not the deltoids. I would submit that the explanation of these cases could be, not that the pectoralis major (upper part) was unable to advance the arms and cross them, but that the first case was similar to those I have described, and the second case was probably a myopathy in which the pectorals were affected, but not the deltoids sufficiently to prevent them crossing the arms.

5. What is the diagnostic value of paralysis in a muscle for one movement and not for another?

This question follows almost as a corollary to the preceding one. It will be obvious, I think, that this condition of a muscle or group of muscles can only be brought about by a central lesion, as it would be difficult to imagine that a disease of the muscle fibres, such as occurs in pseudo-hypertrophic paralysis and other myopathics, or a neuritis of the peripheral nerve, between the brachial plexus and the muscle, could affect one class of movements of a muscle more than another; we should expect them to be diminished or paralysed equally. With regard to central diseases, I am not aware at present of any case of disease of the cortex cerebri or pyramidal tract in which the lesion was so localised as to cause paralysis of the muscles elevating the shoulder and not those depressing it, but it is in diseases of the spinal cord that the condition is best shown. Cases of polio-myelitis and amyotrophic lateral sclerosis have already been mentioned, and I have also seen cases of infantile paralysis and progressive muscular atrophy shewing the same condition. Though I have not had the opportunity of examining a case of injury to the cervical roots, before they join the plexus, I think we are justified in assuming, from the resemblance of the grouping of the paralysed muscles with those due to central spinal disease, that the same condition would be found.

The position of the trophic centres in the brachial enlargement of the cord for the clavicular fibres of the pectoralis major is an interesting question in connection with the second of my cases related above, *i.e.*, that of the boy; for although the muscle was paralysed for movements associated with the deltoid, its trophic centre was not sufficiently affected to give any reaction of degeneration with either form of electricity. Whether the trophic centre for the clavicular fibres is in quite the same part of the cord as for the sternal fibres seems doubtful from a case of infantile paralysis, which I have previously observed and recorded, where the sternal fibres were completely absent, and yet the clavicular fibres were well developed and reacted normally to both currents.[1] Further observations will probably throw more light on this point.

From the above considerations I think we have another symptom to enable us to diagnose on the one hand, between central disease affecting the spinal cord and on the other, lesions affecting the peripheral nerves and the various myopathies, while the condition of a muscle being paralysed for one movement and not for another is in favour of the groupings of the muscles in the brachial and lumbar enlargements being rather of a physiological than of a purely anatomical nature.

In conclusion I would submit that:—

1. Examination of the muscles, when they are visible, in contraction on the living body gives the most exact results as to their actions.

2. The antagonists of a muscle cannot be observed to contract in violent voluntary movements; in slow moderate movements it is doubtful if the antagonists do more than moderate the fall of the limb by gravity; while in very fine exact movements it is probable that both sets of muscles act together.

3. As far as we know there is no instance of a muscle producing an action diametrically opposite to its usual action.

4. A muscle of the limbs can be paralysed for one kind of movement, and not for another.

5. And this condition would point to the lesion being in the nuclei of the spinal cord or its roots, and not in the peripheral nerves or the muscles primarily.

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[1] Cases illustrating localisation in the spinal cord, Med. Chir. Trans., 1885.

URIC ACID IN DISEASES OF THE NERVOUS SYSTEM.

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SIR A. GARROD says ("Gout and Rheumatic Gout," ed. iii., p. 275): "The causes which predispose to gout, independently of those connected with individual peculiarity, are either such as produce an increased formation of uric acid in the system, or lead to its retention in the blood."

My researches for reasons given elsewhere have led me to disbelieve in formation and to credit all uric acid diseases to the last-mentioned cause—its retention in the blood and tissues.

My problem, therefore, in this paper is to trace as far as I can the influence on the nervous system of the uric acid thus retained; and I would remind those who may not have read my previous papers on the subject that I am able to influence the excretion of uric acid at will, and can store up and retain it if need be in the blood and tissues.

As a rule, however, nature has already produced sufficient retention for all purposes, and we are more often called upon to eliminate what has been stored in the body than to produce retention.

I mention the fact, however, to show that I am not speaking of any theoretical process, but of one that can be easily and has been repeatedly demonstrated.

Murchison, in his Croonian Lectures (1874), introduced the term lithæmia to indicate a morbid state of the blood, due, in his opinion, to the abnormal disintegration of albuminous matter in the liver, bringing about an *excessive production* of uric acid and an excess of urate in the urine (lithuria).

I am in complete accord with Murchison as to the symptoms of the condition which he describes, but I differ from him in thinking that the liver symptoms are secondary or concomitant, and are the results of excess of uric acid, rather than the signs of its formation, and in believing that the excess of uric acid in the blood is the result of failure to excrete the amount which is formed in about the normal relation to urea of 1—33.

As I have said elsewhere, if only a fraction more than 1 gr. of uric acid is held back every day we can account for 1 oz. in a year, and that alone would furnish enough for many pathological processes.

Murchison mentions that Austin Flint suggests the term uricæmia for the condition, and quite recently Von Jaksch in publishing his results (*Deutsche Med. Wochenschr.* No. 53, p. 741) uses the term "uric-acidæmie."

For my part I prefer either of these latter terms to that of Murchison, which has been associated for some time with what I regard as an erroneous assumption, and probably the uric-acidæmia of Von Jaksch is least liable to be mistaken.[1]

A considerable number of diseases of the nervous system have been associated by numerous writers more or less directly with gout, or as by Murchison with lithæmia, and my researches have led me to go a step further, and assert that these diseases are, in many cases, not only associated with gout, but are absolutely due to uric acidæmia.

That this being so it is in my power to control the excretion of uric acid and the amount in the blood, and in so doing to produce or prevent several of the diseases in question which are present with uric acidæmia, but depart and remain absent with its removal, so that I have been led in some cases to assert that if the uric acid is attended to the nerve system will take care of itself.

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[1] With regard to the theory of uric acid formation, I see that Dr. Carter Gray *New York Medical Journal*, Jan., 1886) says, in speaking of Murchison's explanation, "Nor does the assertion that the excessive formation of uric acid is due to functional disease of the liver rise above the dignity of a pure assumption."

My impression is that the first point to inquire into is whether there ever is any excessive formation of uric acid, and whether that also is anything more than a pure assumption. I believe that if the matter were thoroughly enquired into it would appear that there is excess of uric acid in the blood and urine to-day, because some four or five grains of uric acid formed several days ago, failed to pass at once out of the body, and are now trying to find their way out. Such at least is the verdict of my researches, in which I sometimes estimated the whole excretion of uric acid for six to eight months and more, without an interval (see Wood's "Medical and Surgical Monograph," New York, Feb., 1890, p. 318).

I propose in this paper to give a sketch of the process by which I arrived at this position, and as far as may be in short space to set out the reasons for my assertions, which represent after all but a small step in a direction to which the work of many observers had long been pointing.

The disorders of the nervous system which Murchison associated with lithæmia are ("Functional Derangements of the Liver," p. 105) :—

1. Aching pains in the limbs, and lassitude.
2. Pain in the shoulder.
3. Hepatic neuralgia.
4. Severe cramp in legs, &c.
5. Headache.
6. Vertigo and temporary dimness of sight.
7. Convulsions.
8. Paralysis.
9. Noises in the ears.
10. Sleeplessness.
11. Depression of spirits.
12. Irritability of temper.
13. Cerebral symptoms and the typhoid state.

Of some of these, as 5, 6, 7, 10, 11, and 12, I shall have a great deal to say presently; 1 and 11 are common in uric acidæmia; 2 and 3 may both point to gastro-hepatic derangement, such as I have often seen with the same blood condition.

With regard to 4, one of the most severe attacks of cramp in the legs I have ever seen was in the person of a school friend, who subsequently had two attacks of acute rheumatism with some cardiac damage, and I have attempted to show that acute rheumatism is due to uric acid. (See *Med. Chi. Trans.*, vol. lxxiii., p. 297, and *Practitioner*, Feb., 1891).

As to 5, Dr. Murchison appears to draw a distinction between the headache of lithæmia and migraine, though he afterwards says, "Megrim, as Dr. Liveing states, is sometimes the expression of what is called a latent gouty diathesis, or in other words, of lithæmia," and he then quotes Trousseau to the effect that migraine is "a manifestation of the gouty diathesis."

About 8 I have some experience, and should add on to it aphasia, about which I shall speak later.

No. 9 and other ear symptoms I have mentioned in previous papers, and about 13 I hope to learn some more when some of the other conditions have been worked out further.

I was myself a sufferer from migraine, or as I now call it uric acid headache, and it was in trying to find out something about the causation and treatment of my trouble that I began this research.

The strong affinity of migraine to gout noticed by Liveing and others led me to examine the urine, and after working for a considerable time with no apparent result I at last landed upon the fact that when the urine excreted during a headache is carefully separated from that excreted before the headache began, or after it left off, an excess of uric acid relative to urea is always found (say the relation 1—20 or 1—25).

Before the headache begins there is often a relation of 1—40, *i.e.*, diminished uric acid, and after it ends the same. If we have a mixture of before or after with the headache urine, the excess in one direction may balance that in the other, and the result comes out near 1—33, or normal; these facts completely accounting for the negative result I obtained at the beginning of my research before I recognised the importance of separating carefully the urine of the headache.

I published my results in a paper before the Royal Medical and Chirurgical Society (see *Transactions,*[1] vol. lxx.), and I have since found just the same thing in a large number of other cases.

Having obtained then this apparent connection in time between the occurrence of the headache and the excretion of uric acid in the urine, I was still in the dark as to its exact meaning.

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[1] "The Relation of a Certain Form of Headache to the Excretion of Uric Acid."

While looking about for a clue to this I noticed the great importance which was attached by Sir A. Garrod to the action of beer and wine in producing gout, and having examined several specimens of beer and found that they were very strongly acid (see *Medico-Chirurgical Transactions*, previous reference), and also that they had a rather marked effect both on headache and the mental condition, it occurred to me to try the effects on the excretion of uric acid of acids direct, and I found, to my surprise, that by means of acids on the one side and alkalies on the other I was able to alter the excretion of uric acid in either direction at pleasure. These results were published in the *Journal of Physiology*, vol. viii., Nos. 3 and 4:—"Variations in the Excretion of Uric Acid produced by Administration of Acids and Alkalies."

After this I soon found out that in influencing the excretion of uric acid I could influence the headache also, that acids which diminished the excretion of uric acid in the urine relieved or removed the headache, while on the other hand alkalies increased the excretion of uric acid and made the headache worse.

I also found that by retaining or storing up uric acid for a day or two by means of acids, I could accumulate it in the body, and by then giving an alkali I could bring out a lot of it into the blood and urine, and could produce the headache at will, and proved at the same time that the headache is proportional to the excess of uric acid over urea, and not to the amount of alkali used to bring it out.

Since that I have also been able to show that if uric acid is previously removed by a course of salicylate of soda, a dose of alkali will produce neither a plus excretion of uric acid nor a headache, so that in every way the headache is proportional to the amount of uric acid in the urine, and I have inferred from this that the amount in the urine may, to some extent, be regarded as an index of the amount in the blood, though more direct proof of this fact can now, I think, be given (see St. Bartholomew's Hosp. Reports, vol. xxvi.).

The close clinical relationship of migraine to epilepsy traced by Liveing and others (see my paper, "Some Clinical Features of the Uric Acid Headache," St. Bartholomew's Hospital Reports, vol. xxiii.) led me to look for a similar excretion of uric acid in epilepsy, and the results in one of the first cases I was able to examine with sufficient care is reported in my paper, "Beitrag zu der Beziehung zwischen gewissen Formen von epilepsie und der Auss-cheidung von Haruäsure" (*Neurologisches Centralblatt*, Mar. 1888), and here again I have since found the same relation in numerous other cases.

About this time the intimate relation of mental depression on the one hand and mental exaltation or well-being on the other to uric acid was forced on my notice.

For in addition to my own experience that mental depression accompanied the uric acid headache, it was observed by myself and others that the period of headache and mental depression was often closely preceded by a period of mental exaltation and well-being, but I have mentioned above that the excretion of uric acid is diminished before the headache; therefore the mental condition varies directly with the relative amount of uric acid in the urine, and I was not long in putting this point to the same experimental tests I had used in the case of headache, and I published a paper on "Mental Depression and the Excretion of Uric Acid" in the *Practitioner*, Nov. 1888.

Here also there came out another important parallel between migraine and epilepsy, for I found that so great an authority as Dr. Ross ("Diseases of the Nervous System," vol. ii., p. 916, 1st ed.) had recorded a precisely similar period of mental exaltation as preceding, in some cases, the onset of epileptic fits, and I had found (see previous reference) that in epilepsy, just as in migraine, the excretion of uric acid is greatly diminished before the attack—that is, mental exaltation corresponds to a minus excretion of uric acid and headache, an epileptic fit or mental depression corresponds to a plus excretion, which is, to some extent, the result of the previous minus excretion (retention).

We shall see, I think, presently what is the probable explanation of this connection, but the facts themselves are of great interest apart from their causation.

Once more a prominent symptom in the attack of migraine gave me a clue to what I believe to be a further advance, and one which I expect will eventually be seen to be more important than all the others combined.

The intense and persistent coldness of the hands and feet in a migraine attack had impressed themselves on me long before I began to study medicine, and when I knew more about it I could not fail to observe what had often been recorded by others (Dr. Liveing "Megrim and Sick Headache," p. 329) that the pulse was slow and the vessel hard and full between the beats during the attack.

And a similar course of experimentation soon showed me that in altering the excretion of uric acid I altered not only the mental condition or the headache, but also the rate and tension of the pulse, and I put forward in the *British Medical Journal*[1] the thesis "that, *cæteris paribus*, arterial tension varies with the amount of uric acid that is circulating in the blood."

Since that time I have accumulated many hundreds of pulse traces, which I think prove absolutely that the knowledge thus obtained gives me practically complete power over the rate and tension of the pulse, which in the great majority of cases is under the influence of uric acid in one way or the other. With my own pulse I can practically do anything I please, and in some diseases with slow pulses I have been able to almost double the rate while reducing the tension, with drugs affecting the uric acid.

I mention this here because the influence of uric acid on the vessels bears importantly on what I have to say as to the possible cause of some of the above nervous diseases; but I am still investigating many matters concerning the effect of uric acid on the pulse, and shall publish my results in due course.

Further, I must qualify my above quotation from the *Brit. Med. Journ*,[1] so far as to say that arterial tension varies with the amount of uric acid in the blood when combined with alkali (soda or potash), as is generally the case in nature, but is little or at all affected by it, even though present in considerable quantity, if combined with a salicylate (salicyluric acid).

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[1] February, 1889.

So much for the history of my work up to the present time, the results of which I shall now proceed to apply to the etiology of certain functional nervous disorders ; but will first of all point out that I have been previously led to suggest that opium produces its well-known effects on the mental condition, for the sake of which it is so often taken to excess, through its effect on the circulation of the brain, which is secondary to its effect on uric acid, which in turn depends on its influence on the alkalinity of the blood and so on the solubility of uric acid.

The matter may be shortly stated as follows : all substances which increase the solubility of uric acid in the blood increase its excretion in the urine, and conversely all substances which diminish the solubility of uric acid in the blood dimish its excretion in the urine, and with certain reservations, which need not now detain us, an excess of uric acid in the urine may be looked upon as the index of some excess in the blood.

The chief substances which diminish the excretion of uric acid in the urine are acids, iron and lead, and those which increase it, soda or potash under certain conditions, phosphate of soda and salicylates (*Medico-Chirurgical Trans.*, vol. lxxi. and lxxii.) Opium must be added to the first group, because, as I have pointed out (*Brit. Med. Journ.*, Nov., 1889.[1]), it raises the acidity of the urine, diminishes the alkalinity of the blood, and consequently reduces the amount of uric acid it can contain. Hence like acids, iron or lead (*Medico-Chi. Trans.*, vol. lxxi., p. 283), it relaxes the arterioles and improves the circulation in the brain, producing mental brightness and well being.

Mercury is another instance of the same thing: it markedly reduces the excretion of uric acid in the urine;

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[1] "Influence of Opium and Morphine on Uric Acid."

that it markedly reduces the tension of the pulse and produces diuresis has been pointed out by others, and I have suggested that these results are entirely due to its effects on uric acid, as similar results can be produced by all drugs which influence uric acid in the same way and to the same extent. (See *Brit. Med. Journ.*, June, 1890-1241. "Causation of reduced Arterial Tension by Mercury.")

With regard to the causation of the headache of uric acidæmia there is the theory of Du Bois Raymond, quoted by Liveing, that migraine is due to a tetanus in the muscular walls of the vessel of the affected part of the head, and Dr. Brunton[1] has suggested that the proximal part of a vessel may be dilated while the distal part is contracted, and that the impulse of the blood against the contracted portion may cause pain.

For my part I know that some cases of migraine (I believe a large number) are contemporaneous with an excessive excretion of uric acid and its excess in the blood; I also know that uric acid in the blood contracts the arterioles and capillaries all over the body, producing the cold surface and extremities I have noticed, raising the tension of the pulse, and, in accordance with Marey's law, that pulse rate varies inversely as arterial tension, slowing the heart; hence, I have no difficulty in believing that uric acid can produce one of the above-mentioned conditions, and I look upon migraine, or the headache of uric acidæmia, as a local vascular effect of uric acid.

I have said that I can produce or remove it at pleasure by acting on uric acid, but that when there is no uric acid, drugs alone will not produce it; I can also show in my own case and that of others, that a diet in which milk, with occasional very small quantities of fish or egg, constitute the only animal food, will completely prevent attacks, and that without any drugs whatever to affect the nervous system.

The effect of this diet is, first of all, to reduce considerably the *formation* of uric acid and urea (which, however, continue to be formed in their natural relative proportion, 1—33), and, secondly, with the lessened formation of urea there is a lessened formation of acid, so that the supply of alkali in the blood is good, and the solubility of uric acid is rarely interfered with. Hence nearly all the uric acid formed on this diet is freely excreted and at once got rid of.

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[1] "Disorders of Digestion," p. 103.

I believe that a man eating meat three times a day and drinking acid beer or wine may keep back and store up in his body several ounces of uric acid in a few years, while on the above diet he would not store as many grains.

I have been on such a diet now for a good many years (at least seven or eight), and I almost never have a headache; but if the headache was due to alkali, for instance, it should be much worse, as the acidity of my urine is now lower and the alkalinity of my blood greater than when on ordinary diet.

On the other hand, if I infringe my rules and eat meat and drink wine for two or three days in any single week I am very promptly reminded of my infirmities, and suffer with a bad headache.

I say therefore most positively with regard to this headache, look after the uric acid and the nervous system will look after itself.

Epilepsy resembles migraine in the mental brightness and well being, with scanty excretion of uric acid, which may precede both, in the excessive excretion of uric acid and mental depression which may accompany both, and in the subnormal surface temperature which is often found with both; and as we shall see the pulse in some cases of epilepsy is slow and faltering, as it often is when the tension is high. There are further points of resemblance in the facts that both may come on early in life and last for years or the whole of life, that both tend to recur at more or less regular intervals; both are often met with in members of the same family, or both may affect one and the same patient—now a fit, now a headache, or a fit may come in the middle of a headache. Two cases in my paper ("Some Clinical Features of the Uric Acid Headache," St. Bartholomew Hospital Reports, vol. xxiii.) suffered from both fits and headaches, and fairly numerous cases of the same thing have been recorded by others, and in my experience epilepsy and headache, gout or rheumatism, are very commonly met with in the same family.

In drug action, again, there is a certain amount of parallelism; acids, and many drugs which act as acids, as nitro-glycerine, and nitrite of amyl, relieve both.

Opium relieves migraine, but insures its return next day, unless the uric acid is meanwhile removed by a salicylate, when the opium rebound is, as I have shown, to a large extent robbed of its horrors, and this has led me to suggest that in chronic morphinism (see paper on opium, previous reference) a course of salicylate might give great help in the attempt to break through the habit.

Opium, it will be remembered, has been used with success in some puerperal and uræmic convulsions, but I have not had much experience of it in epilepsy, though I have occasionally used it in place of acids or nitrites.

Iron often does harm in migraine, and lead, which has a similar action on uric acid, is an accredited cause of convulsions; and both, no doubt, act by retaining the uric acid on one day, and laying up a store for the production of uric acidæmia on a subsequent day, when fits result.

Quinine causes headache in some people, and is said by others ("Quain's" Dictionary, p. 450) to do harm in epilepsy; I can show that in certain conditions connected with enlargement of the spleen or ague it may bring a very large amount of uric acid into the blood, and then I should expect it to do much harm in both disorders.

It is interesting to note in this connection that Dr. L. C. Gray (*New York Medical Journal*, January, 1886) considers that malaria produces lithæmia; thus he says, "Malaria, as I have said, often precedes lithæmia, and apparently causes or excites it, but the distinction should be sharply made between the two, because the quinine which may alone cure the former generally aggravates the latter."

Both these observations I can explain and confirm. Ague no doubt acts like influenza and other fevers, of which I shall speak presently, in storing up uric acid in the body; if it also causes an enlargement of the spleen it provides an additional storehouse for uric acid, and when the fever ends and the acidity falls some of these stores are from time to time washed out into the blood, causing uric acidæmia, and if quinine is now given it increases the outflow of the uric acid (? by contracting the spleen) and the amount in the blood, aggravating the lithæmia as Dr. Gray observes.

Closely related both to migraine and convulsions is mental depression, dulness, and inability for mental effort and bad temper, and as a matter of fact I rarely see the one without enquiring for the other two.

Since I first wrote on uric acid and mental depression I have heard from Professor C. Lange, of Copenhagen, that he has for a long time observed, and has written a monograph[1] concerning certain conditions of periodical depression which he connects with the uric acid diathesis, and treats successfully by a diminution of meat food, and certain rules as to exercise, &c.

I have also previously mentioned that Dr. Broadbent notices the frequent association of melancholia and mental depression with high tension of the pulse, and records the successful treatment of some of these cases by milk diet.

These facts help us to explain many points in connection with functional disorders of the brain, some of which I have already alluded to. Mental irritability and depression and their results, murder and suicide, are perhaps the most interesting of these. In several previous papers[2] I have spoken of the way in which the excretion of uric acid may explain the excess of suicides in July. As my researches show that there is an excessive excretion of uric acid in the warm months of the year, especially in July, and conversely a minus excretion in the colder months, especially January; during the plus excretion in July there will be relatively high arterial tension with anæmia of the brain and mental depression, and this coincides with my own experience, as I suffer much more from mental depression in the warmer months of the year than at any other time, and as the mental depression accounts for the suicides, so the irritability and bad temper which accompany and are due to the anæmia of the brain, will, I have no doubt, explain the excess of murders in certain months—thus May has been called the month of suicides and murders (*Lancet*, Vol. I., 1890, p. 1208), and it is not difficult to explain this, for May is practically the first month in which we have any warmth after a long period of cold, and there will consequently, at this time, be a large amount of uric acid in store, ready to be excreted, as the acidity falls for the first time with returning warmth, and I think that if two curves were made, one showing the temperature and the other the number of suicides, they would be found to correspond pretty closely, especially at the early part of the warm season up to July.

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[1] "Om Periodiske Depressionstilstande," Cobenhavn, 1886.

[2] *Practitioner*, Nov., 1888, and Wood's "Monographs," Feb., 1890, prev. ref.

So that I have come to believe that many a case of suicide and perhaps some murders also might be prevented by a timely dose of acid, which would free the brain circulation from the power of the uric acid, and produce (as shown by Drs. Roy and Sherrington, *Journal of Physiology*, vol. xi., p. 85) an increase in its size and a free flow of blood from its vessels.

Further, some facts which have recently come out have too important a bearing on this subject to be passed over. I refer to the remarkable increase of suicide that followed in the wake of the epidemic of influenza. I noticed it myself, having seen an unusually large number of suicides and cases of mental depression in February and March, 1890, and as I was myself a sufferer from the epidemic I was able to follow out the changes in uric acid excretion with great accuracy.

And what I found was this, that as in the case of other fevers, the rise of temperature at the onset entailed (see paper in Virchow's *Archiv* of June 1, 1889, p. 337, by Dr. Peiper, who says that the alkalescence of the blood is diminished in all fevers) a rise in the acidity of the urine and a fall in the alkalinity of the blood and tissue fluids, this driving the uric acid out of these fluids and the blood (as I have shown elsewhere) diminishing its excretion in the urine and retaining it in the body, say for the sake of definiteness in the liver, spleen and joints, though some of those attacked by influenza were able to say very definitely where the uric acid had gone to, from the muscular and articular pains experienced. (See paper on "Causation of Acute Rheumatism," read at Med. Chi. Soc., April 22, 1890, and *Practitioner*, Feb., 1891.)

We have, then, a sudden sharp fever causing retention of uric acid in the body to the extent of a considerable number of grains, and coming in this country at a time of year (January) when there had been already from other causes (cold season, previously mentioned) some retention of uric acid in the body.

We have every reason to think, then, that the store of uric acid in the body at the end of the febrile period of influenza would be very large; as the fever declines and is followed by debility and dyspepsia, often with pulmonary catarrh, and other results of the infection, the acidity rapidly falls, the metabolism of the body slackens, and the excretion of urea falls off by as much as 100 grains. Then as the acidity of the urine falls and the alkalinity of the blood and tissue fluids rises, the stored uric acid is acted upon, and it is washed out from its hiding place. There is at once a plus excretion of uric acid in the urine and an excess of it in the blood, producing the circulatory effects of which I have been speaking.

Then the excretion of uric acid will be great in proportion to the amount that has been stored in the body, and this will be large in proportion to the severity of the fever, the diet of the patient, and the amount which he had previously stored before the fever began, and it seems probable that the storage will be greater in the case of a fever like influenza, which supervenes suddenly on a condition of ordinary health, as compared with that in a fever like typhoid, which comes on gradually with some failure of nutrition, and a plus excretion rather than a retention of uric acid.

And I have seen far too many cases of the same thing in other sufferers to believe that we are not dealing with cause and effect, and in those cases that have come to me for mental depression, provided there was no organic disease of heart, vessels, kidneys or brain, I have had no hesitation about promising complete relief to the symptoms.

With regard to suicide I was much interested in a paper read by Dr. Bertillon, before the Epidemiological Society (see *Lancet*, Jan., 1890, p. 1019), in which he mentioned that the suicides in France had increased 40 per cent. after the influenza epidemic—a fact, he remarked, which he was quite unable to account for, but which to my mind presents very little difficulty, and might have been foreseen. I will conclude by remarking that a similar train of symptoms and results is to be seen after other fevers, as pneumonia, rheumatic fever, scarlet fever, no doubt due to the same cause, *i.e.*, temporary excess of uric acid in the blood from the store retained by the high acidity of the fever, and that this fact accounts to my mind, as I shall have to point out at some future time, for the relapse of rheumatic fever, which indeed can be produced at pleasure (see previous reference to Med. Chi. paper), and for the rheumatic symptoms and possibly the nephritis which so often follow scarlet fever (see paper, "On the Connecting Link Between the High Tension Pulse and Albuminuria," *British Medical Journal*, Jan., 1890, p. 65).

Another instance of the same thing is no doubt to be seen in the frequency of suicide during the menstrual epoch in females which is alluded to by Dr. Barnes in a letter to the *British Medical Journal* on the relations of the female sexual organs to mental diseases (*Journal*, Vol. I., p. 1401), 1890.

There is, I think, no doubt whatever that the menstrual molimen is accompanied by a certain amount of digestive disturbance which brings about a temporary fall in acidity and thus determines a plus excretion of uric acid and its temporary excess in the blood, and this in turn acts on the vessels and the cerebral circulation in the way I have been describing, and brings about cerebral anæmia, mental depression and suicide.

In complete accordance with this is the fact that the pulse tension is generally high during the menstrual period, and it may be remembered also that uric acid headache and the fits of epilepsy are generally to be met with at the menstrual period of those women who suffer from them.

On these points Dr. Broadbent says ("The Pulse," p. 76)

"It is in the early morning that depression of spirits is liable to be at its worst in nervous debility, so-called, or there is the morning headache which is relieved by the bath and breakfast, or wears off as the day advances, or the subject of this affection is more tired on waking up than on going to bed. In heart disease, again, the sufferer will wake up gasping for breath after sleep, and paroxysms of asthma and the fits in epilepsy choose this time for coming on."

I shall have no difficulty in proving that it is just in these early morning hours that the excess of uric acid in the blood is greatest, and its effects on arterial tension most marked, so that it is very easy to explain the causation of the conditions mentioned by Dr. Broadbent.

I have already frequently pointed out that the excretion of uric acid in the urine and its amount in the blood vary inversely as the acidity of the urine, and directly as the alkalinity of the blood.

There will therefore be most uric acid in the blood when its alkalinity is highest and the acidity of the urine lowest, and it may be seen from my curve in Wood's "Monographs" (prev. ref., p. 319) that at nine a.m. the acidity of the urine is less than at any other hour in the day, and I have other curves and observations which show that this low acidity begins about six a.m. or sooner, and the reason of this is not far to seek.

The acid tide of the night reaches its height about midnight or in the small hours, and then from lack of supplies begins to fall away, and by five, six, or seven a.m. has run down very much.

During the period of high acidity the excretion of uric acid is small, and its amount in the blood minute; it is all driven into other quarters where, if it is present in any quantity, it makes itself felt in increase of gouty and rheumatic pains; at this period there is low pulse tension, with quick pulse and a very copious excretion of urine—in fact the largest hourly excretion in the twenty-four hours. But as the acid tide period comes to an end, and the acidity of urine falls, all this is reversed; the uric acid is brought out from its various places of retention by a fresh supply of the solvent alkali, the amount in the blood quickly arises till it may contain almost as much as is usually present in a dilute urine, and with this the arterioles and capillaries contract, the tension rises and the heart slows, the urine becomes scanty, and when the hours of sleep are coming to an end there are heaviness, drowsiness, great disinclination to turn out, and a feeling that one is in more real need of sleep now than at the beginning of the night's rest. All this takes place every day as regularly as the sun goes round, and disease takes its origin in a mere intensification of a process which is natural and physiological.

The high tension pulse of the early morning hours is of great interest, and I have many traces which show it, some taken on purpose before leaving my bed, and these show clearly enough, though the tension is commonly sufficiently obvious to the finger, that all the warmth of the blankets (and where is one warmer than in bed?) is powerless to completely relax arterioles which are contracted by uric acid.

In accordance with this the urine is often very scanty at this time, as 35 to 40 cc. in the hour, giving at the same rate only 840 to 960 cc. in the twenty-four hours, and in my paper on the action of mercury on arterial tension (*British Medical Journal*, June, 1890, p. 1243) I have mentioned that the excretion was one morning only 53 cc. in the hour when the tension was high, and that not because there was no fluid in the body to be excreted, but because the arterioles of the kidney were contracted by uric acid, as I was able, by taking a dose of opium in solid form to relax the arterioles, and run the excretion up to 234 cc. in the following hour.

Such fluctuations in the excretion of uric acid and its amount in the blood occur every day, and it is only when they are exaggerated by errors in diet, by exposure to weather or the action of disease, such as influenza, mumps, pneumonia, scarlatina, that their effects may be serious, and pass over into the region of pathology.

More than that I have, as previously stated, absolute control over the excretion of uric acid and the amount of it in the blood, and can either increase or intensify these natural fluctuations or may act in opposition to them and prevent their appearance at the usual time. On the one hand by diminishing the alkalinity of the blood I can free it from uric acid, relax the arterioles, cause pricking and shooting pains in the joints, or greatly increase any previous gouty or rheumatic pains, quicken the pulse and produce mental happiness and well being, relieving headache or mental depression if previously present. On the other hand, by increasing the alkalinity of the blood I can cause it to be more or less flooded with uric acid, can slow the pulse, contract the arterioles and raise the arterial tension, greatly diminish the excretion of urine and relieve the joint pains, producing in their place, however, a sluggish circulation in the brain with general languor, depression and disinclination for exertion, and if there is much uric acid, perhaps even headache or a fit. On the other hand, if I have previously taken the precaution of removing all the available uric acid from the body by a course of salicylate, I can give as much alkali as I like without producing any of those effects, and, conversely, if I have carefully collected uric acid for some days previously by means of acids, all the effects of giving an alkali are intensified. This proving to my mind conclusively that the effects in question are due to the uric acid and not to the drugs, which only act indirectly.

This series of events may be repeated to any extent, and I should be sorry to state the number of times I have produced them or watched them in myself and others.

We can now, I think, completely understand the meaning of Dr. Broadbent's observation—why depression and headache are worse in the morning, why some are more tired then, why cases of morbus cordis wake up gasping, and why asthma (see Wood's "Monographs," p. 359) and epilepsy (see previous remarks) choose this time for coming on.

I have long ago inferred that just as the uric acid headache is due to excess of uric acid in the blood and its effects on the arterioles of the brain, so its nearest relative epilepsy is also in many cases due to disturbance of function having a similar origin.

But it is now, I think, quite possible to go further and assert not only that uric acid is the cause, but also to point out with a considerable degree of certainty its actual *modus operandi.*

In his extremely interesting book on "The Pulse," Dr. Broadbent, in speaking of Tripier's theories about the pulse of epilepsy, says (p. 120), "I think it is the slowing of the circulation which causes the convulsions," and on p. 122 he says, "I look upon convulsive attacks, when they occur in connection with an infrequent pulse, as a result of cerebral anæmia, produced exactly in the same way as the convulsions after great hæmorrhage." And further on he remarks, "The late Dr. Moxon, it will be remembered, went so far as to suggest that the initial event in common epilepsy was a stoppage of the heart."

Now it seems to me that we have in the effects of uric acid on the circulation of which I have spoken a direct cause of just such alterations in the cerebral circulation as Dr. Broadbent here refers to, and its seems probable that uric acid may produce these results in two ways: (1) by contracting the cerebral vessels and producing anæmia of the brain with probable stasis in certain portions of it; or (2) by causing such a general rise of arterial tension that the heart falters and flutters, the pulse gets slower and slower, and finally, for a beat or two, stops, and down goes the patient in a faint with or without anæmic convulsions, and this latter case explains probably Dr. Moxon's suggestion alluded to by Dr. Broadbent.

I have pointed out that there is excess of uric acid in the urine and probably in the blood at the time of the fit, and have suggested that this has a causative relation to the attacks just as in its near relative and neighbour the uric acid headache.

I would point out also in passing that the plus tension produced by uric acid accounts for the slowing of the pulse, in accordance with Marey's law previously mentioned.

It thus seems to me that there is every reason to believe that uric acid, by contracting the arterioles and capillaries, may produce what is practically stasis in certain areas of the cerebral circulation, this being the exciting cause of a fit;

under slightly differing conditions, whether in the amount of uric acid present or in the arrangement and distribution of the cerebral vessels, it may only produce sluggish circulation and anæmia, these accounting for mental depression or a headache.

Hysteria and hystero-epilepsy are, I have no doubt, related conditions due to the same causes, and I have seen attacks of this nature supervene in the middle of what appeared to be any ordinary uric acid headache, with plus excretion of uric acid in the urine. Again, Dr. Broadbent has pointed out that the pulse in hysteria commonly shows high tension ("The Pulse," p. 275).

So that I should regard hysteria as one of the mental effects of uric acid, and as a variety or mixed condition between ordinary epilepsy and simple mental depression.

Nor need we, I think, stop here with the causation of (? functional) epilepsy, for it seems equally probable that the effect of uric acid on the arterioles may precipitate fits in cases where they are clearly due to an organic cause, as, for instance, in the common association of a series of epileptic attacks with syphilitic disease, new growth or accidental injury of bone or membrane.

The new growth is there constantly and may, we will suppose, be steadily enlarging, and yet the fits are occasional, not constant; it seems to me that the vascular effects of uric acid may have their point of greatest action determined by the tumour, and that the fits may be really due to uric acid acting on a vascular area of the brain already hampered by the presence of a new growth; the immediate effects of the new growth on the circulation of adjacent parts of the brain may be looked upon as the charge of powder, while the uric acid and its vascular effects act the part of the percussion cap in starting the explosion.

I should not, however, agree with Dr. Broadbent that the high arterial tension is "probably the most important factor in the secretion of the pale and watery urine which accompanies an hysterical attack." As a matter of fact, during the high arterial tension and contraction of arterioles and capillaries in the attack, the urine is scanty and of high specific gravity, containing much uric acid; and it is only when the tension falls and the capillaries are relaxed at the end of the attack that it becomes pale and watery.

Before leaving the causation of fits, I will just mention that on p. 245 Dr. Broadbent gives what seem to me to be very good reasons for believing that the fits and other phenomena of uræmia are not due, as was supposed, to the direct action of the poison on the brain, but rather to anæmia of, or stasis in the brain, these being the consequence of high tension, which again is secondary to the poison in the blood, and the contraction of the capillaries it produces.

The interesting bearing of all this on much that I have previously said must be obvious, and in uræmia we have evidently to deal with a poison which, if not uric acid, at least affects the capillaries in exactly the same way that uric acid does, and has, as I may point out at some future time, many other points of resemblance to it; and I have already pointed out that opium probably relieves some cases of uræmia and puerperal convulsions by acting on uric acid (*British Medical Journal*, November 1889).[1]

There is another point with regard to epilepsy which I think uric acid will explain, and that is the drowsiness and stupor which commonly follow the fits. This has, I think, been generally said to be due to exhaustion of the nerve centres after the discharge of the fit, but if this is its causation it ought, to some extent at least, to be proportional to the severity of the fit, so that I was much interested to see that Dr. W. S. Colman (*Lancet*, 2, 1890, p. 14) relates that this is not always the case; thus he says, "In some, for example, the convulsion may be severe, and the recovery of consciousness rapid, while in others, although the convulsion may be slight, the patient may remain in a state of unconsciousness for some time."

From my point of view there is very little difficulty in explaining these facts, for I take it that the stupor or unconsciousness which follows the fit is just as much due to the action of uric acid on the circulation of the brain as the fit itself, and it depends upon the rate at which the uric acid is driven out of the circulation, and its effects on the vessels neutralised, how long the condition will last.

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[1] See also my paper in St. Bartholomew's Hospital Reports, vol. xxvi., for Von Jaksch's results, showing excess of uric acid in the blood in nephritis, and my explanation of the fact.

The fluctuations of uric acid vary almost infinitely; as times one meets with a large amount in the blood, but present for a short time only, at others a smaller amount continuing to be present for a longer time; in the former case the fit may be severe, but not followed by much unconsciousness; in the latter there may be hardly sufficient uric acid to produce a fit at all, but yet the drowsiness and stupor may last a long time.

In accordance with this reasoning I may say that I have been for a considerable time in the habit of giving such cases of epilepsy as come under my care a dose of acid to take as soon as they can be got to swallow anything after the attack, and in a very considerable number of cases the friends have told me that they come round more quickly out of the stupor when this acid is given, and in one case the patient used to make signs that he wanted the dose as soon as consciousness began to return. I suppose he found that it relieved his head.

Now the effect of an acid given in this way would be to hasten the expulsion of uric acid from the blood, and the delivery of the cerebral circulation from its power.

I have thus come to believe that the unconsciousness which follows the fits will last a long or short time according as the uric acid which occasioned them is slowly or quickly driven out of the blood, and that the stupor is not really due to exhaustion of the nerve centres any more than, as we have seen, the heavy, languid, and sleepy feelings so often met with in the morning hours during the plus excretion of uric acid, are due to want of sleep in the previous night.

I would also remark that the feelings of depression, heaviness and disinclination for bodily and mental exertion which are part of the mental depression of uric acid, and which, to a large extent, can be either produced or removed at pleasure by influencing the uric acid, differ from the stupor which follows an epileptic fit in degree only.

And further, it is in my experience no very uncommon thing for a certain amount of drowsiness, disinclination for exertion, mental or bodily, or even a headache, to precede the onset of an epileptic fit, and I believe that if these things were inquired for they would be more often found by others. Certainly a considerable number of epileptics have told me that they are more heavy and drowsy on waking on those mornings on which a fit subsequently occurs, and we have seen the explanation of this. We shall also see that the scanty urine during the fit and the free flow which follows it are evidences of a similar and contemporaneous action of uric acid on the vessels of the kidneys. When these vessels are contracted the urine is scanty and of high specific gravity, and the subsequent dilatation which frees the brain circulation produces a copious flow from the kidney.

It is, I think, an almost necessary sequence of what I have been saying, that uric acid by its influence on the arterioles will affect portions of the nervous system other than the brain in the same way, and when it affects the cord may, with or without the co-operation of cardiac failure, produce conditions of paresis or even of paraplegia. Similarly also uric acid may affect the peripheral nerves either indirectly through affecting the arterioles which nourish them, or more directly by its influence on the fibrous tissues which support them, just as it affects similar tissues in other positions in the body; and in reference to some such effects I have several times noticed what appears to be a fairly constant relation between the amount of uric acid in the blood and the presence, absence or alteration in amount of certain forms of tremor, but I shall have more to say on these subjects at some future time.

I will now just say one word about aphasia which interests me very greatly, as I myself suffer from it very markedly at times of excess of uric acid in the blood, *i.e.*, when there is mental depression, lethargy and threatening or presence of slight headache.

I have mentioned it before in speaking of mental depression (see *Practitioner*, Nov., 1888, p. 344), but this matter has, I think, recently gained fresh interest from the very able lecture of Dr. Bastian on the subject (*Lancet*, Vol. I., 1890, p. 1164). As he says there in a sentence which interests me very much, "Thus where there is a slight lowering of functional activity in the auditory centre we have produced a slight amnesia with forgetfulness of names of persons and things, the centre not being sufficiently active in this condition to respond to some volitional and associational incitations." Now this is exactly the condition which I have previously described as occurring in myself and occurring under just those conditions in which, from what I have previously said, we should expect the circulation in certain regions of the brain to be hampered by uric acid, this resulting in a lowering of their functional activity. Further, I know as a fact that my father suffered from time to time for a large part of his life from just such functional asphasia, and eventually in old age he had organic asphasia with right hemiplegia, from which he never completely recovered, though he lived for twelve years. May we infer from these facts that the hæmorrhage which caused the organic aphasia had its situation determined, to some extent, by the changes produced in the vessels and nervous structures of the affected region by long years of recurring disturbance of function, and eventually of nutrition, by uric acid? May we also infer that I have inherited some peculiarities in vessel structure and distribution which predispose exactly the same regions in my brain to react in the same way to an excess of uric acid in the blood?

With regard to aphasia Dr. Broadbent says (p. 249), "There may, for example, be fugitive aphasia or amblyopia or hemiopia, which are easily explained by stasis or ischæmia, in certain vascular areas, but incomprehensible as the results of a poison circulating everywhere through the brain," and with regard to the retinal circulation and the possible causation of ischæmia or thrombosis in it by uric acid, I have been specially interested in a paper in the *Ophthalmic Review*, March, 1890, by Mr. Stanford Morton, as he narrates cases where repeated paroxysmal failure of sight accompanied migraine and high tension of the pulse, and mentions that in some instances the arterial contraction was sufficiently long continued for thrombosis to take place with permanent loss of sight. Here, then, we have visibly going on in the retina in company with migraine and high arterial tension, just such changes as occurring in other invisible regions of the brain may be the cause of the fits in epilepsy, the mental depression or melancholia and the headache, all of which have a similar relation to uric acid and the high tension it produces, and I have great hopes that examination of the retina under various, if necessary artificially produced, changes in the arterial tension may throw much light on the exact mode of causation of some of these diseases.

In my paper on mental depression I noted that several cases of melancholia or depression had been observed to clear up suddenly on the supervention of an attack of gout.

The cause (rise of acidity) that drove the uric acid out of the blood into the joint relieved the cerebral circulation from its effects, and it seems to me that it would be a rational treatment of such cases to produce an attack of gout, which is sometimes easy, but if there was great debility or cyanosis it might present considerable difficulty.

I have also observed a considerable amount of depression and tendency to suicide in cases of nephritis, and if they suffer from chronic excess of uric acid in their blood[1] there is no difficulty in accounting for either the high tension pulse or the depression, and in those cases where I have been able to lower arterial tension and quicken the pulse I have also markedly improved the mental condition.

But uric acidæmia affecting the arterioles and capillaries may have other important relations to the nutrition and structure of the nerve centres.

In speaking of the causation of epilepsy I suggested that some fits were really of the nature of syncope, the heart being overpowered by the capillary contraction of uric acidæmia, and I believe that in some cases of morbus cordis a final stop may be put to the heart's action by a sudden increase of the uric acid in circulation, and in some hearts that are not diseased or even weak, very serious functional disturbance, almost going the length of syncope, may be produced.

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[1] See Von Jaksch, prev. ref. Also Dr. Alice Bennett, Procs. of Med. Socy. of Pennsylvania, June, 1890, for some interesting cases.

I have a good many traces showing intermission of my own pulse, but out of many hundreds of such traces I have none showing intermission which do not also show high tension, so that I regard the intermission of my pulse as a flutter on the part of a heart which is temporarily overpowered in the struggle with the arterio-capillary contraction produced by uric acidæmia.

And, obviously, if it is completely overpowered we shall have, more or less, prolonged syncope, which may be fatal.

Now, if syncope is, to any large extent, due to the cause of which I am speaking, it ought to be most often met with at those hours of the day at which uric acid is generally present in some excess in the blood, viz., the three or four hours immediately following the morning meal (in some cases, especially with gastric disturbance, an hour before breakfast also, see previous remarks), and a second period in the afternoon, generally from three to six p.m.

And I would ask those who take an interest in this matter to make a note of the time of occurrence of the cases of syncope they may see.

The time relation would probably show best in those cases occurring unexpectedly in people previously in apparent health, for where there is disease and malnutrition or dyspepsia, uric acid excretion and its passage through the blood is subject to all kinds of variations as to time of day.

Another effect of high tension is probably to be seen in rupture of a vessel, as in cerebral hæmorrhage, and here, again, the trouble should be more frequent at the hours mentioned above, and I have records of several cases in which the rupture has occurred in the alkaline tide period.

And if when uric acidæmia is contracting the capillaries and raising the tension, the heart is hypertrophied and does not give way or falter, it may be that one of the vessels will yield on the proximal side of the obstruction and hæmorrhage result.

With regard to the causation of syncope by uric acid, the newspaper account of the death of the late Canon Liddon interested me very greatly; supposing that account to be true, it is almost certainly an instance in point.

In the *Standard*, Sept. 10th, 1890, it is recorded that "In July, after taking cold, he was reported to be suffering from gout in the head and lying at Christ Church, Oxford;" from this he got better, and "on Friday last he was removed to Weston-super-Mare. The journey exhausted him for a time . . . . but he was able to enjoy a carriage drive on each of the three following days. On Monday (8th Sept.) he remarked that he had entirely recovered from the weakness following upon the railway journey"—this indicating, I should say, a relative feeling of well being, with rise of acidity and retention of uric acid.

Next morning (Tuesday, 9th September) "he partook of breakfast with Mrs. King and her daughters, and made no complaint of feeling any worse than usual," and then mark what follows, the uric acid retained on the previous day all rushed out in the alkaline tide after breakfast, the resulting uric acidæmia contracted the arterioles, overpowered the heart and produced fatal syncope. "Shortly after the meal, however, Canon Liddon was found in a fainting condition," and death was attributed to "failure of the heart's action."

With regard to the vertigo of uric acidæmia, Dr. Buzzard, in a most interesting address before the Harveian Society "On Vertigo of Bulbar Origin" (*Lancet*, Jan., 1890, p. 179), mentions that a gouty history is very common in these cases, that some have high tension of pulse, and suggests that their trouble "may be due to the presence in the blood of uric acid salt or some equivalent," and he also appears to have found salicylate of soda useful in treatment, the explanation of which he kindly credits to my researches.

It appears to me practically certain that if uric acid affects the arterioles and the cerebral circulation in the way I have attempted to sketch out, a very slight change in the position of its maximum effect will account for the production of vertigo; and it may be of interest if I mention here that I have myself suffered from at least one well-marked attack of this trouble, which came on one morning just as I was thinking of turning out of bed, and was followed by vomiting; and what I have previously said about the time of causation of headache and fits will apply to this, which I look upon as an accidental variation of my migraine.

I am also interested in the large discharge of colourless urine noticed by Dr. Buzzard as following the attacks in some cases, and I should refer it to the same causes that produce the diuresis following migraine, hysteria or epilepsy, viz., to the relaxation of the capillaries of the kidney at the end of the attack, as the blood was cleared of the uric acid which had produced their contraction; and I find in many cases, as I have said, that just as I can control the rate and tension of the pulse, the headache and the mental depression, so I can also control the flow of urine.

Sleeplessness is now about the only thing on Dr. Murchison's list which I have not spoken of. I have little to say about it, except to endorse most fully Dr. Broadbent's teaching that in the sleeplessness of high tension a dose of mercury is the best treatment ("The Pulse," p. 278-9).

I have no practical doubt that such high tension and sleeplessness is due to uric acidæmia, and that the mercury acts in the way I have pointed out upon the uric acid.

I see that Dr. Tyson ("A Clinical Note on the Soporific Action of Mercury" *Brit. Med. Journal*, vol. i., 1891, p. 221) speaks of the kind of condition in which he has noticed this action of mercury as one of "biliousness," and yet further on he distinctly says that he does not associate these cases with "lithæmia."

I can only say that I have gradually grown to look upon what is commonly called a bilious attack as equivalent to a uric acid storm with uric acidæmia, and I have no practical doubt that mercury relieves the sleeplessness of this condition by acting on uric acid in the way I have pointed out (see previous reference).

I must say a few words on diagnosis of the above disdisturbances of the nervous system as the result of uric acidæmia, lest it should be said that I claim all forms of headache, epilepsy, and mental depression as due to uric acid, which is very far from being the case.

As regards the uric acid headache, the chief points are that it is periodical, that it comes once in every seven, ten, fourteen, or thirty days for years or for life, often beginning in childhood, that it lasts one day or less, rarely two days, and that it tends to be worst at those hours at which the excretion of uric acid is normally greatest.

I have seen headaches which bore a superficial resemblance to it, but differed in lasting practically without intermission for seven to ten days; this I think should put the ordinary headache of uric acidæmia out of the question, and search should be made for organic disease or nephritis, though I am not prepared to say that the headache of nephritis is not in some cases due to chronic uric acidæmia, but then the uric acidæmia in this case is probably due to an organic and not to a functional cause.

The family history, again, is often very characteristic, many members suffering from headache often called "bilious," with a history of gout, rheumatism, and not very rarely, phthisis.

Last, but not least, there are the slow high tension pulse and cold extremities, the uric acid reaction in the urine which I have described, and the often very decisive effects of drugs and diet.

Mental depression due to uric acidæmia is often periodical, as pointed out by Prof. C. Lange; it tends to be worse just at the same hours that the headache does (alkaline tide), and is always markedly worse in the warm months of the year as compared with the cold ones, for the reasons previously gone into, and I have also mentioned its relation to acute diseases. Where depression is continuous and varies but little from hour to hour or day to day, morbus cordis nephritis or other organic disease should be carefully looked for. The pulse is slow and of high tension during the depression.

With epilepsy perhaps the first thing to do is to exclude obvious organic, specific, or reflex cause, then if the family history of gout and uric acidæmia is fairly strong a urinary reaction may be looked for, and if obtained the case may be further tested as to the effects of diet and drugs.

In examining the urine the utmost care must be taken to separate the urine of the attack from that before and after, or a negative result will be obtained, which, however, cannot be held to prove anything, just as my first negative results in the case of the uric acid headache proved nothing.

The rate and tension of the pulse is probably just as important as in the case of the headache and mental depression of uric acidæmia; it also seems to me that the attacks are more common at those hours of the day at which the amount of uric acid in the blood is naturally greatest, and in several cases I have been distinctly told that attacks were more frequent in the summer than in the winter, thus corresponding exactly with what occurs in mental depression and the excretion of uric acid.

I must also say a few words about treatment, as it is often necessary to try the effects of treatment to clinch the diagnosis.

And first as to uric acidæmia in general, I will state shortly what I believe to be the mode of production of an excess of uric acid in the body. From my point of view the chief or only cause is deficient elimination of uric acid in the urine, which is generally the result of high acidity interfering with its solubility.

This is the first stage of deficient excretion holding back or retention of uric acid in the body; it may be looked upon as going on to some extent in every one during the best years of nutrition and bodily activity, and it will be great in proportion to the excess of nitrogen taken in and the excess of acidity from the salts of meat and the acids of beer and wine.

I take it, then, that at fifty years of age every Englishman has accumulated in this way a considerable store of uric in his body, and it can be found at this time in large quantity in the liver, spleen, and many other tissues, but up to this time he has but rarely had any excess in his blood, his high acidity and vigorous nutrition have kept that fluid clear.

Now, however, the scene changes, he has several hundred grains of uric acid on board; what will he do with it? or rather, what will it do with him?

During the next ten years there is almost always some failure of nutrition, exercise is deficient, digestion is impaired, food is not properly digested and absorbed, and with these changes and in consequence of them, there is a fall in nitrogenous metabolism, a fall in urea, and still more important, a fall in acidity.

Now comes the turn of the stored uric acid; with the fall of acidity the solvent power of the blood is increased, and so long as the stores last the blood will be flooded with it, and more or less chronic uric acidæmia marks the declining years of life, with excessive excretion in the urine as its sign.

This explains what appears to have been a great stumbling-block to some observers, why patients at this time of life if put on a farinaceous diet go on excreting excess of uric acid for months or even years; of course they will do so, and must do so, till their stores and accumulations are exhausted.

The great cause of uric acidæmia then is: 1st, the accumulation of uric acid in the body; 2nd, the appearance on the scene of a supply of alkali to bring it into solution in the blood, such supply of alkali being met with in nature generally as the result of some failure of nutrition.

The indications then are clear but vary somewhat according to the stage at which the process has arrived.

In young people, for instance, whose stores of uric acid are probably small but who suffer from periodical uric acidæmia due to fluctuations in excretion, the fluctuations accounting for the periodicity of their headache, mental depression, or fits, as I have pointed out before, it may be enough to alter the diet and reduce the nitrogenous metabolism while providing for the continued free excretion of all the uric acid formed; but in older men it will be necessary to clear out the stores that have accumulated in past years as well as to reduce formation, and the clearing out is probably best effected by a course of salicylate of soda, continued if necessary for weeks or months, till the daily excretion of uric acid remains for some time at the level of formation (1—33), and this being accomplished the nitrogenous intake should be reduced to the lowest level compatible with healthy nutrition. If in place of a salicylate you give an alkali to aid the removal of the accumulated uric acid you will produce intense uric acidæmia and precipitate in one form or another the troubles we are anxious to avoid.

A uric acid headache, when present, may be treated with a dose of any acid which, when it raises the acidity of the urine and diminishes the alkalinity of the blood, will rid the latter of uric acid and put an end to the trouble; but it may fail to raise the acidity of the urine, either because it is not absorbed owing to a sick stomach, or because it is overpowered by a large amount of alkali present, or coming from other sources—as food previously taken.

What is to be done then? If there is a sick stomach I usually attack it locally with a mustard-leaf and try spiritus ammoniæ aromat. or nux vomica in place of acids; sometimes opium or mercury in small dose will succeed where acids fail. They all, as I have previously pointed out, act in the same way by clearing the blood (and consequently the urine) of uric acid.

Having thus cleared the blood of the uric acid give salicylate of soda for two or three days to sweep it out of the body and get rid of it, but never give the salicylate while the headache is bad, it will increase the gastric trouble, cause or increase nausea, gastric absorption will fail altogether for a time even if there is no vomiting, and the headache will be much worse than if you had done nothing; the best rule is to give acids first and get the stomach into good humour and then give salicylates.

Warm clothing and hot water to the feet generally do good by counteracting the contraction of the arterioles to some extent.

But prevention is far more satisfactory than treatment of the attack by drugs, though individual attacks can be controlled by attending to the points I have mentioned.

In my own case two or three meals with meat and wine in one week will almost certainly produce a headache, proving that my nerve centres are as sensitive to the vascular effects of uric acid as ever, but that when I do what is necessary to keep them free from uric acid they are otherwise able to perform their functions.

Occasionally when a little forethought is exercised the bad effects of indiscretion in diet can be prevented by taking salicylate of soda for a day or two afterwards.

The treatment of epilepsy is a much more difficult matter, and even if it were possible to prove absolutely that the fits in any given case are due to uric acidæmia their treatment or prevention would still be a very difficult matter.

As I have said, I can practically produce or remove the headache of uric acidæmia at pleasure, and anyone can tell when they have got a headache and when it is gone ; but supposing that in the same way I was to produce a fit it would be almost impossible to prove that I had produced it ; or if I put a stop to a series of fits it would be impossible to prove that they might not have stopped if I had done nothing.

By following the uric acid from hour to hour the onset of a fit might be foretold, or the same might sometimes be done from observation of the pulse tension, headache, drowsiness, or depression ; and if these symptoms were accompanied by fits when we did nothing, but were not followed by fits when we acted on the uric acid and removed them, we might reasonably claim that we had prevented a fit, but such argument is not very convincing.

In my experience the drugs that are useful in the headache of uric acidæmia are useful also in the fits, a series of fits corresponding, as I believe, to a headache and being often produced by the same cause, namely, some gastric upset from fatigue, cold, inhalation of chloroform, &c., which causes the blood to be flooded with uric acid.

In some cases acids do good, in others opium or nitrites, occasionally a course of salicylate of soda seems to cause marked improvement, especially if it is given along with an acid mixture in alternate doses.

But after all said and done drugs can never master the uric acid, it is sure to slip past you at one time or other, even if your drugs do not, as in the case of headache, occasionally interfere with digestion and nutrition, and make matters worse.

Diet seems to me the only hope, and even from this I expect far less than in the case of headache, especially when the fits have gone on for a long time—the more often the trigger is pulled the slighter the touch required to fire the charge; even if the nutrition of the involved structures is not eventually impaired in the way I have suggested above when speaking of aphasia. It is on record, however, that diet has done good in a considerable number of cases of epilepsy, and Dr. Broadbent has pointed out (Croonian Lectures, 1887) that cases of epilepsy in which there is high tension of the pulse are especially benefited by a farinaceous diet. (See also L. C. Gray, *New York Medical Journal*, July, 1884.)

I have not myself seen a case of epilepsy in which this diet has been given what I should regard as a fair and satisfactory trial; with the poor the difficulty too often is that they can't, and with the rich that they won't, and imperfect half-hearted trials are not of much value.

Meanwhile I look upon a distinct connection with uric acidæmia in any case as an indication for such a diet, and hope to meet with some cases in which it can be done as thoroughly for epilepsy as in my own case for headache, and possibly a diet in which milk is the only animal food may sometimes be necessary in epilepsy.

The treatment of the mental depression of uric acidæmia presents no special points; once the diagnosis is complete it is that of uric acidæmia above given: 1st—Remove the stores of uric acid, if any, and then get on to diet, and here again Prof. Lange and Dr. Broadbent have recorded good results from a milk or farinaceous diet. In slight cases due to temporary causes a course of acids and salicylates will sometimes work wonders.

With regard to the effect of acids in mental depression I will just relate one fact which seems to me of interest as tending to show how general and widespread is the action of uric acid on the mental condition.

In a review of some of my writings in *Prager Med. Wochenschr.*, 1889, No. 28, Dr. G. Hofmann mentions, in reference to the effects of acids on the mental condition as given in my paper in the *Practitioner* (previous reference), that in Western Bohemia the good effect of acids on the mental condition has passed into a proverb, which he quotes.

And since I have come to understand the excretion of uric acid and the influence of meals upon it, especially its excessive excretion in certain hours of the morning and afternoon with a corresponding temporary uric acidæmia, I have been able to recognise the results of this uric acidæmia in myself and others to a much greater extent than was previously possible, and I have no doubt that the man who falls asleep over his paper after breakfast, or yawns and stretches and is unfit for good mental work for an hour or more after lunch, is not suffering from dyspepsia but from uric acidæmia, though of course if he has dyspepsia the uric acidæmia will be increased in consequence of it.

In conclusion, I will say that I look upon these functional disorders as the result of the vascular conditions produced by uric acidæmia, the exact nature of the disorder—whether headache, epilepsy, or mental depression—being determined partly by the intensity of the uric acidæmia and partly by the anatomical and physiological relations of the vessels and nerve structures concerned, and that these latter, being inherited, determine in several succeeding generations the nature of the disturbance which uric acidæmia will produce.

That the uric acid and its excretion is in no way dependent on the nerve centre, but that if the uric acid is removed its vascular effects and their secondary results will disappear, provided that during the action of the uric acidæmia no lesion of structure has been brought about.

On the other hand I quite believe that the structure of the nerve centres and the distribution of its vessels not only determines the kind of disturbance which uric acidæmia will produce in any given case, but also explains why one individual suffers in this way from functional nervous disorders, while another with about as much uric acid in his body and blood escapes. (The escape is, however, only relative, in so far as the symptoms are too slight to attract attention, but the action of uric acid on the vessels is, I believe, universal).

I am also aware that when the nervous system is depressed by fatigue, deficient food, &c., a smaller amount of uric acid in the blood will suffice to produce disturbance of function than at other times; but nevertheless it is practically true that if uric acidæmia is prevented the nervous system will not itself originate these disturbances, and that this knowledge of the effects of uric acidæmia gives complete power to produce or remove the vascular conditions, and the nervous disorders, which are secondary to these conditions, are to a large extent also within our power.

Clinical Cases.

KATATONIE. SEQUEL OF A CASE. NECROPSY.

BY W. JULIUS MICKLE, M.D., F.R.C.P. (LOND.).

It may be of interest to complete a case already reported, by the necropsy notes in abstract. The clinical features and history of this case were described by me in this journal.[1] That report carried the case to the time it was written, September 1889. Subsequently, the patient at times would querulously stop one to demand satisfaction as to his release, and would declare that "a fine game" was being played with him. His statements were stereotyped. Now and then he refused food for a meal or so, on some of these occasions alleging that he had not any appetite, on others that the food was not good enough for him.

He fell ill and died during the influenza epidemic here in January, 1890, but co-existent muco-colitis yielded part of the symptoms and part of the lesions observed. Preceded by some anorexia and slight vomiting, this acute illness began on the morning of the 21st. Shortly after taking part of some fluid food, he rapidly became bluish and livid in the face, and had dyspnœa, with a noisy respiration of 33, a pulse of 96; pain in the head, shoulders, chest and sides of trunk, but chiefly in the chest; cold feet, rigors. He was put to bed, hot bottles were applied to the feet, and a mixture of brandy and carbonate of ammonia was given in small repeated doses. A watery fluid and the food taken were vomited. And now for a time the lips and face were livid, and hands purple; the pulse varied from 128 to 118 per minute; but the respiration sank successively to 30, to 22 and to 18; the intense pain in the chest was relieved, and for the time the facial lividity nearly disappeared. The heart sounds and respiratory sounds were enfeebled, and the pulse was almost imperceptible at wrist. Temp. 104·5°. Hst. quiniæ, grs. iv. At noon he felt "ill all over;" had nausea and pain in the head; the breathing was too audible, but was only 15; the pulse was 120. There was cough, with watery frothy expectoration. The worst pain was opposite the middle of the sternum. Over the lower and posterior surfaces of the lungs were small dry *râles*. To be sponged every hour with tepid water and have enemata of peptonized milk every three hours. P.m. temp., 103·5°. During the night had eight muco-sanguineous stools.

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[1] See Brain, 1889, vol. xii., p. 503.

22*nd*.—A.m. pulse, 115, resp. 30, temp. 102·5°. Is taking a little egg and milk, and stimulating mixture, and now and then a nutritive enema. Still has some muco-sanguineous stools. Abdomen poulticed; two draughts, each of grs. iv. of quinine; tepid sponging all over every hour. P.m. pulse 118, resp. 36, temp. 103·2°.

23*rd*.—Still inclined to vomit; with difficulty induced to take milk, egg and coffee. It taking small doses of tr. opii and vin. ipecac. Pulse 120, resp. 42—later on 52, temp. 101·5°. Hands livid, purplish; face dusky and livid; pulse almost imperceptible; tendency to cardiac and respiratory paralysis; heart and lung sounds feeble; pupils dilated. To take brandy, carbonate of ammonia and sp. ætheris. P.m. pulse 135, resp. 54, temp. 98·9°. Takes milk and above mixtures. Has throughout the illness had special attendants day and night. Still complains of pain at chest and has dysphagia. During illness has had and still has many perversions of sensibility, especially of taste, and delusions with regard to what is given him; *e.g.*, he says milk is not milk at all, nor is anything offered to him accepted as what it purports to be, but is purposely tampered with, he alleges. His chief sufferings have been from dyspnœa and respiratory and cardiac oppression, and lividity has been marked nearly throughout. Latterly, the legs have become mottled with purplish patches, and the hands cold and clammy. Gradually becoming worse, he dies early on the morning of the 24th.

In the following necropsy-record the principal noteworthy points are:—

(1) The deviations from normal type, or atypical peculiarities, of cerebral fissures and gyri, in relation to the patient's criminal and insane antecedents, described in the former communication to this journal.

In relation to katatonic: (2) the existence, at the anterior part of the inferior and mesial surfaces of the cerebral hemispheres, of patches of what I[1] have termed "adhesion and decortication;" namely, adhesion of pia to brain and separation of superficial layers of the grey when the membranes are stripped off. Also (3) the slight thickening and opacity of the pia-arachnoid over the brain base; and great thickening, toughness and opacity over the supero-lateral, fronto-parietal region, with corresponding pial œdema, over-easy removal of meninges, and wasting of gyri. Also (4) the cortical microscopical appearances.

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[1] *Journal of Mental Science*, January, 1876, and April, 1878.

(5) The brain generally firmish. The brain also congested at least by the final illness and mode of dying; and some evidence of old hyperæmia, &c.

(6) The thick heavy calvaria of rounded triangular shape in transverse section, the rounded apex representing the forehead.

Necropsy fifty-eight hours after death.—Well-nourished; rigor mortis; much purplish hypostatic lividity; also irregular, purplish mottled discoloration of the limbs.

Head.—The posterior part of calvaria is the widest, the parieta eminences being very far back, and immediately behind them the steep occipital declivity; thus the cross-section of the calvaria has the shape of a triangle with rounded angles, the apex in front. At all parts of the cross-section the calvaria is thick, especially in the frontal region and in the posterior parietal; the thickening affects both tables and the diplöe. The last-named is congested. The skull is fairly symmetrical on the two sides; the weight of the portion removed at usual level is 17 ozs.

The dura-mater is slightly thickened; in the falx cerebri is a limpet-shell-like osteoma; the lateral dural sinuses are full of dark blood and clot. The dura lining the front and middle regions of the base is somewhat thickened, and has a slight rusty stain on the free surface. The arachnoid is thickened over the interpeduncular space, and over the orbital surface is somewhat opaque, thickened, and tough. There is some wasting of the olfactory bulbs and tracts, and part of the former remain adherent to the skull when the brain is removed. Over the tip of each frontal lobe is a bone-like plate in the pia-arachnoid, oblong, thin, and in area about equal to a circle of $\frac{1}{4}$ or $\frac{1}{3}$ inch diameter. A third similar, and similarly situate, but smaller, plate is also found near the tip of the left frontal. There are slight adhesions between the two frontal lobes; and some adhesion and decortication (see below). Considerable opacity of arachnoid over upper and lateral aspects of frontal and parietal regions, ending pretty sharply opposite the parieto-occipital fissure. The same tract of the pia-arachnoid is considerably thickened, tough, and leathery, and separates with too much ease from the surface, a little watery fluid draining away from the sulci as the tunics are peeled off. These changes are chiefly marked over the middle of the lateral region and posterior part of parietal. The pia is congested; the meningeal veins are much congested, especially behind. These meningeal changes are fairly symmetrical on the two sides. Although nearly free from these changes, the occipital meninges separate with abnormal ease.

The sulci are somewhat rounded, and the convolutions somewhat wasted in the supero-lateral fronto-parietal regions of the cerebrum. In the cerebral cortex, the grey matter is of fair depth, shows some vessel-contents visible to the naked eye, is of fair colour and stratification, and of fair consistence, or perhaps very slightly of increased consistence (considering the interval since death). The grey matter of orbital surface is perhaps slightly bluer and redder than that of upper surface. In the right hemisphere there is some adhesion and decortication over the anterior curve of the gyrus fornicatus, and over the marginal gyrus adjoining the lower part of this, and over the back part of the gyrus rectus, and over part of the insula. On the left side, the gyrus rectus is similarly affected at its back part, and the gyrus fornicatus and insula are affected similarly in kind, but much more slightly in extent and degree. In the white cerebral substance are—stringy clot in the vessels, numerous puncta cruenta, and a congested appearance; the consistence firmish. Basal ganglia firmish and of fair vascularity. Ependyma of lateral ventricles well defined; some distension of Galen's veins; velum interpositum thick and tough.

Cerebellum.—Meninges firm over its inferior surface, and when being cut resist toughly. Cerebellar substance slightly firmish, and, relatively to cerebrum, a little pale. Ependyma of fourth ventricle slightly indurate and opaque. Pons and medulla oblongata firmish, of pale pink hue. Weights: entire encephalon, &c., as removed, 52 ozs.; right cerebral hemisphere 22 ozs., left ditto $21\frac{5}{8}$, cerebellum $5\frac{1}{2}$ ozs.; pons and medulla oblongata just under 1 oz.

Microscopic.—Back part of left superior frontal gyrus. The specimen was kept too long in spirit and weak chromic acid solution, and some of the changes may be artefact.

The bases, especially of many of the cortical nerve-cells, are surrounded by vacuoles; this affects large and medium-sized cells. Many of the cells, perhaps especially in the fusiform-cell layer, are dull, opaque, having most of the protoplasm so highly stained with aniline blue-black that the nucleus is obscured, and the cells get a dark and very slightly and factitiously quasi-granular appearance. Other cells show a large, more deeply-stained nucleus.

At parts, especially in the outer two layers, are vacuoles due to empty cellular spaces, roundish holes existing there in the sections. A few of the capillary vessels show increased nuclei of their walls. Around some vessels is a very distinct perivascular space, which in one instance is partly occupied by nuclear bodies. Several of the blood-vessels are slightly tortuous. Here and there small masses of brownish blood-pigment are very sparsely scattered in the stroma. Nothing markedly altered in white substance of gyrus.

Architectural condition and anomalies. As to the condition and anomalies of the brain-architecture in the right cerebral hemisphere.—Fissure of Rolando very zigzag. Præcentral fissure runs from close to the great longitudinal fissure, to be superficially confluent with the Sylvian, and gives off a branch which becomes superficially confluent with Rolando's. The perpendicular limb of the Sylvian is confluent with the second frontal sulcus. The interparietal fissure is confluent with the Sylvian, and with the transverse occipital sulcus, and is bridged across from the supramarginal gyrus. Horizontal limb of Sylvian fissure is unusually short. The external limb of the parieto-occipital fissure is bifurcate, the anterior branch cutting off a peninsula consisting of the upper and posterior angle of the quadrate lobule (a condition I have found in many brains of the insane). The posterior termination of the calloso-marginal fissure is much hooked and bifurcate, the posterior fork joining with præcuneal sulci, and zigzaging across the præcuneus nearly to the parieto-occipital fissure. The supra-marginal gyrus is subdivided into two tiers by unusual tertiary sulci. First temporo-sphenoidal sulcus well-marked; the second badly marked, indistinct, atypically divided into segments.

On both sides the calcarine fissure is not confluent with the hippocampal. In both hemispheres the occipital lobes are short and small.

Left cerebral hemisphere.—The third frontal convolution is far more normal in its relations on this than on the right side, but its posterior extremity is slightly sunken and concealed. The præcentral sulcus and the perpendicular limb of the Sylvian have the usual form and relations. The interparietal fissure, confluent with Sylvian and with transverse occipital sulcus, is very zigzag, and, on this side, not bridged. The Rolandic runs into the great longitudinal fissure. On this side the parieto-occipital fissure is of usual form and relations.

Thorax, Heart.—Right cardiac chambers filled with dark fluid blood and clot. Dark, mingled with firm pale, clot in pulmonary arteries. Firm, pale pre-mortem clot in, and adherent to walls of, right ventricle; and in pulmonary veins. Opacities in coronary arteries, aortic sinuses of Valsalva, and anterior flap of mitral valve. Heart-muscle unduly friable. Weight 6½ ozs.

Tracheal mucosa reddened and swollen, and also is that of larynx above the vocal cords.

Left lung.—Bronchial mucosa reddened, lined with a little blood-stained secretion, and swollen. These changes extend more or less to the minute bronchioles. Venous blood exudes from the surface of sections of the lung. The postero-inferior portion of the lung is darker than the rest, and less crepitant, and is slightly both collapsed and congested. Weight 15¼ ozs.

Right lung.—Some old pleuritic adhesions at posterior part of apex; old scars and slight traces of former tubercle at apex. In the posterior part of lower lobe are patches of collapse, and of congestion and œdema. Weight 18½ ozs.

Abdomen.—*Spleen*, firm, of chocolate hue, 6¼ ozs.; slight oval capsular thickening, and pigmented spots. *Left kidney*, 5 ozs., capsule separates fairly. *Right kidney*, 5¾ ozs., surface mottled by patchy congestion. Liver, centres of acini congested; gall bladder full of dark treacle-like bile.

In the *colon* are scattered hyperæmic, swollen, dark-red patches of the mucosa, here and there denuded of epithelium, or partly so. Some of these small areas are darker: and of deep dark-reddish hue, as a background, covered with a delicate film of ashen colour; and here the surface is slightly roughened by the epithelial alteration, destruction, and shedding. These changes are well-marked in the transverse colon, are much the same in its descending portion, but less in the ascending.

CASE OF TABES DORSALIS.

BY J. MICHELL CLARKE, M.A., M.B., M.R.C.P.,
Assistant Lecturer on Physiology, Bristol Medical School; Assistant Physician General Hospital.

In the following case of Tabes Dorsalis, the presence of nystagmus, the persistence in some degree of the knee-jerks, and the predominance of symptoms in the upper extremities afford unusual features in the course of this disease. The patient, a warehouseman, aged fifty-three, first came under my observation on October 15th, 1888, and has been constantly under my care since. Except that his mother suffered from asthma, that two brothers died in infancy, and that one sister has dropsy of cardiac origin, his family history gave us no information. No member of his family had ever suffered from any nervous disease, nor from gout nor rheumatism, his grandparents on both sides dying at an advanced age. He had always been very healthy until four years previously, when he had a severe fall, breaking his right leg. To this fall he attributes his illness, as he says, that "it shook his nerves all over." He is a very temperate man, and has never had syphilis.

Careful enquiry, however, elicited that for a year before the fall he had suffered from burning pains in the arms and hands, and this must be therefore regarded as the first symptom of the disease, the course of which was probably accelerated by the accident. After the injury he suffered from general weakness and numbness of arms and legs, with well marked girdle pain, and "rheumatic gout" in right great toe joint, and from shooting pains in arms and legs, followed by a feeling as if cold water was being poured over him. For two years he had been giddy, especially in the dark, with a tendency to fall on closing his eyes; for past ten months there had been diplopia. He had not had attacks of palpitation, nor of sickness; micturition was normally performed; habitual constipation; sexual power good.

On examination he is a tall man, and looks older than his years. His gait is feeble and uncertain rather than ataxic, when he tries to walk with his eyes closed he is very unsteady, and there is then some ataxy of movement. He is unable to turn round quickly without catching hold of something to steady himself. He cannot stand steadily with his feet placed together, and Romberg's symptom is well marked.

At this date there was decided want of co-ordination of the movements of the hands and arms with loss of the sense of posture, and of the power of distinguishing between different weights; the inco-ordination increased when the eyes were closed. He had lost the power of writing from inability to hold and direct the pen properly. Sensation to pain, touch and temperature normal, or perhaps slightly deficient. The muscles are rather flabby and weak.

In the legs sensation and muscular sense quite normal; the muscles firm and of good size showed no wasting nor increase of the direct muscular irritability. When lying on the couch he could move his legs freely without ataxy whether the eyes were open or shut. No rigidity of muscles, which everywhere react normally to both kinds of electric current. The right knee-jerk was generally absent, but could sometimes be obtained, the left knee-jerk present but feeble, and could be brought out well by causing him to perform some muscular action, such as hitting out with the left arm, at the same time that the tendon was struck.

The right big toe joint was enlarged, with much bony thickening, and the joint loose and grating, the proper extensor tendon tense, so that the first phalanx was over-extended on the meta tarsal bone, the head of the latter being pushed downwards. In the absence of any family history or personal evidence of gout this was taken to be a tabetic arthropathy. In the eyes there was paralysis of the right external rectus; when the eye was moved into the inner canthus it oscillated, *i.e.*, was jerked two or three times towards the left before remaining steady. The pupils were unequal, the right larger than the left, both acted to accommodation, neither dilated on sensory stimulus to skin of neck, the right was motionless to light, the left acting feebly. No paralysis of any other cranial nerve. Dynamometer gave 30 with right hand, 28 with left.

On one occasion whilst being examined he had a well-marked laryngeal crisis, and during the winter 1888-89 several severe gastric crises. Under the usual treatment, at first with arsenic and iodide of potash, and later with cod-liver oil, quinine, nux-vomica, &c., his symptoms grew steadily worse. From April to November suspension was tried, and was carried out more or less regularly three times a week throughout this period. On account of a tendency to syncope he was never suspended for more than

1½ to 2 minutes at a time. The effect of suspension was seen in marked improvement as regards the legs, he was able to walk more strongly and steadily and for greater distances, the giddiness decreased and he no longer complained of giddiness in the dark; at the end of the time he could stand steadily with the feet together and the eyes closed for five minutes. Since the suspension he has been able to walk about easily, and for considerable distances at a time, and has lost completely the lightning pains in the legs, so that the general effect as regards the legs was very satisfactory. On the other hand the arms grew steadily worse, and he suffered from frequent and severe gastric crises, one lasting for ten days, during which he was exceedingly ill and very weak for some time afterwards. In October, 1889, he had intense burning pains in the arms and severe girdle pain, and in November suspension was left off as the treatment seemed to have accomplished its beneficial action on the leg symptom and not to have affected the arms at all, at any rate in the direction of improvement. Distinct loss of sensation other than subjective feelings of numbness came on in the hands and arms during the summer of 1889, and in November tactile sensation was lost over the right hand, and was very deficient and perverted to temperature (cold objects felt as hot) and to pain. The same loss existed in less degree over the left hand, right forearm and back of left forearm. Except as regards the affection of the upper extremities, which has steadily but slowly increased, his state has remained about the same up to the present time. Lately the gastric crises have ceased, the girdle pain is slight and only occurs occasionally. On examination, February 6th, 1891, he complains chiefly of burning pains in the arms, which are intensified by touching any hot object.

Sensation in Upper Extremities.—Over inner side of left arm the two points of the æsthesiometer are distinguished at a distance of eleven centimetres from each other, but in no other part of arms, forearms, or hands can they be discriminated. A light touch is nowhere felt. Over the right hand he can just tell ice from nearly boiling water; over the arm on the same side sensation to temperature deficient only, but on right forearm and whole of left upper extremity no distinction between hot and cold can be made; cold is felt as hot. As regards pain, sensation is lost over palms and palmar aspects of fingers, and elsewhere is exceedingly defective in both limbs; it is most preserved in the right thumb. He is very slow or fails altogether to localise sensations, and their perception is much delayed. The sense of position of the limbs is entirely lost, so that without sight he cannot say in what position his arms lie. He cannot distinguish between the weight of a shilling and of three sovereigns piled one on the other. Coarse movements are performed by the aid of sight, but very vaguely and imperfectly without it. Finer movements, such as picking up a small object, writing, or buttoning his waistcoat, are impossible. When he tries to slowly touch his nose with his forefinger, the movement is interrupted by ataxic jerkings, apparently with the object of preventing the too rapid movement of his arms. These jerkings sometimes give an appearance of coarse tremor to the movement, and when he holds out his hands, slow movements of extension and flexion of the fingers occur, which resemble athetosis, but are not rhythmic, and evidently result from involuntary irregular relaxations and contractions of the muscles. This may be compared with the great difficulty he has in relaxing a contracted muscle at will. When he sits up and closes his eyes, some tremor of the head and neck becomes obvious.

The muscles generally are weak and flabby, with exaggerated idio-muscular irritability, the pectorals are rather small, and show very well the phenomenon of myoidema. On the other hand, all the tendon reflexes are absent.

Legs and Trunk.—Some defect of sensation to temperature over feet and legs, over thighs and trunk normal; sensation to pain fair, to touch defective over lower extremities generally. Two points are felt as one over the soles of the feet; on the dorsum they are distinguished at a distance apart of 7-8 cc.; on the legs, outer side, 12; inner, 11 cc.; thighs at 4-5 cc., and there is distinct delay in appreciating and localising a touch; over abdomen and back sensation normal, muscles of legs well nourished, movements strong, with little or no inco-ordination, and no loss of muscular sense. The right knee-jerk can occasionally be obtained, but is often absent; the left present, but weak, and can always be brought out if the patient performs a voluntary movement. Tickling the soles of the feet excites a contraction of the anterior muscles of the thigh. Cremasteric, gluteal and plantar reflexes normal. The muscles generally react too readily to percussion on their mass.

Mr. C. H. Walker, the ophthalmic surgeon to the Hospital, reports as the result of his examination of the eyes, that both pupils are slightly irregular in size and shape:—R. 5 mm. diam.; L. 4-5 mm. diam.; R. hazel grey; L. hazel brown in colour. Neither act to accommodation. R., no reaction to light; L. acts to light fairly. In both eyes there is fine lateral nystagmus, which is not constant, but occurs from time to time, and is especially brought out after rotating the eyes several times laterally. There is occasional tremor of the orbicularis palpebrarum—most marked on the left side — coincident with the nystagmus. The left eye is on a somewhat higher level than the right—about 2 mm. higher. The right external rectus is completely paralysed, with corresponding diplopia. The lenses show a few fine cortical opacities. The optic discs are not atrophied, and show no sign of past neuritis, though they are a little irregular in outline. Fields of vision almost normal. Myopic astigmatism of slight degree in both eyes.

The nystagmus was not present when the patient first came under observation ; it was first noticed about eighteen months ago, but as stated above, the right eye from the first, when directed into the inner canthus showed two or three jerking movements before settling down. Possibly, the fine opacities in the lens, acting together, with imperfect co-ordination of the ocular muscles, may account for it, just as nystagmus is often observed in young children who have had corneal opacities from an early age, before the centres for the movements of the eyeballs are fully organized. So that in this case I should conclude that the origin of the nystagmus lay in the imperfect impressions of the muscular sense, reaching the centres from the ocular muscles. together with the presence of opacities on the lens. The movements consist of a very fine and rapid motion horizontally.

A CASE OF TREADLER'S CRAMP.

BY W. H. R. RIVERS, M.D.

J. M., aged fifty-six, was admitted into the National Hospital for the Paralysed and Epileptic on January 30th, 1891, under the care of Dr. Hughlings Jackson, at whose request I publish the case. The patient was shown by Dr. Jackson and myself to a meeting of the Neurological Society on March 5th.

The patient has been a handloom weaver from the age of sixteen. At his work he stands leaning back against a rail, using both hands and working the treadle principally with the right leg. Ten years ago he began to make mistakes in his work, which at first he attributed to some defect in his machine. After a time the right leg became lame in walking, and he then found that the mistakes in his work had been due to imperfect treadling with that leg. The difficulty gradually increased, and he has now been unable to work for five years.

The difficulty occurs at the commencement of the movement of flexion of the hip and knee, which corresponds to the upward motion of the treadle, the extension or downward movement being unaffected; the flexion of the limb when treadling is performed with great difficulty, as if some resistance were being overcome, the thigh becoming inverted during the process.

The spasm can be lessened by supporting the lower end of the thigh, and especially when any pressure is exerted on the popliteal space. Dr. James Taylor informs me that he has observed a similar phenomenon in a case of hammerman's cramp, in which pressure over the median nerve completely abolished the spasm; whether in the present case the improvement is due to any pressure on the nerve I have not been able to determine, but am inclined to attribute it solely to support of the limb.

The patient walks leaning forward and using the right leg very stiffly. He goes upstairs with difficulty; downstairs easily. That the gait should be affected is in accordance with Dr. Poore's observation that, while in an occupation spasm, like writer's cramp, in which the movements concerned are fine, the affection is usually, though not invariably, limited to the act of writing; in those in which the movements are large, as in hammerman's cramp, the limb suffers for modes of action other than that of the occupation. Since both walking and going upstairs involve flexion of the limb similar to that which occurs in treadling, it might be expected that they would suffer in this case.

The left limb is used normally. The tendon jerks are equal and not exaggerated. Certain muscles show decided changes in their electric irritability; the right glutei require a much stronger faradic current than the left, and the right hamstring muscles do not act as readily as those of the opposite side. These muscles show diminished galvanic irritability, but without any qualitative change, and are also wasted as compared with those of the left side. Similar changes in the electrical reactions of the muscles were observed by Dr. Taylor in the case of hammerman's cramp above mentioned.

That there should be these changes in some of the muscles is evidence in favour of the view advanced by Dr. Hughlings Jackson. He considers that the affections known as occupation spasms are due to defective action of some elements of the spinal centres, or their homologues higher up.

Taking the case of writer's cramp, there is, Dr. Jackson supposes, atrophy of a few elements of the anterior horns for the most special movements of writing. In the attempt to write there is over-development of other, more general, movements of the same muscles.

There is, he thinks, paralysis with cramp in at least many of these cases.

Critical Digest.

SEMEIOLOGY OF SLEEP.[1]

BY DR. PAUL BLOCQ.

Chef des travaux Anatomo-Pathologiques à la Salpêtrière.

Translated from the original MS. by H. W. Marett Tims, M.D. (Edin.)

In a large number of maladies, natural sleep presents various modifications, such as insomnia, twitchings and startings, morbid dreams and nightmares, the semeiological value of which we shall first consider.

Again, there exist states of true pathological sleep; hypnotism, vertigo with paralysis, narcolepsy, hysterical sleep, hypnotic sleep, somnambulism—which are more or less analogous to sleep, and of which we shall in the second part point out the differential characteristics.

I.

Insomnia is the more or less complete privation of sleep. As a general rule it depends upon conditions which keep up cerebral excitability. In this respect it is necessary to remember that the duration of natural sleep is variable, according to individual idiosyncracies.

Although insomnia appears in the majority of acute and chronic diseases, it is, in certain cases, none the less a symptom of real importance.

Certain causes, predisposing to insomnia, are well recognised; emotional persons and old men are particularly subject to it; so also, occupations which necessitate a large amount of brain work or compel late hours, would be equally predisposing causes.

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[1] This paper forms part of a treatise on the "Symptomatology of Nervous Diseases," which will shortly be published (in conjunction with T. Onanoff), by M. Masson in Paris.

The determining causes have been divided according as they consist of excitations arising in the nerve centres themselves, or as the result of peripheral impressions transmitted by the nerves; or lastly, as they affect the brain by means of an alteration in the blood.

The majority of affections of the brain itself or of its membranes are causes of insomnia. Nevertheless, some of them only appear to act by the painful sensations to which they give rise; in others, loss of sleep appears as an essential symptom.

Among the former it will be sufficient to mention meningitis, encephalitis, cerebral syphilis and tumours. In the two last cases the insomnia acquires a certain semeiological value, as it is evidence of the intensity of the headache, of its nocturnal character, and may, perhaps, be a guide as to its duration.

Among the latter group one may mention the commencement of a great number of cases of mental alienation, and it is from this point of view, namely of diagnosis, that insomnia is interesting to consider here.

Among the *peripheral excitations*, pain comes in the first rank, whether caused by a wound or neuralgia. A certain number of cutaneous irritations may also be mentioned. All these instances, which it would be wearisome to enumerate, are only important with regard to their differentiation.

Alterations of the blood, which give rise to insomnia, may in the first instance consist only of disorders of the circulation, and from this point insomnia may also be of great diagnostic value, for it is important to know that it is frequently the first sign of an attack of syncope. Alterations of the blood due to infections and intoxications cause sleeplessness. Among the former we may mention, in particular, typhoid fever, of which sleeplessness is often an initial symptom; also acute secondary jaundice is frequently ushered in by insomnia. Among intoxicants it is scarcely necessary to mention coffee and tea.

Lastly, there exists a kind of *essential* insomnia generally, it is true, appearing in those persons with a hereditary nervous temperament in whom it is impossible to point to any direct cause to which one may, with any precision, attribute the trouble.

Twitchings, Startings.—In natural and sound sleep the body is in a state of relaxation, inert, and subject only to the laws of gravity. Nevertheless, by reason of the special mode of cerebral action, which characterises it, the excito-motor power of the cord is increased, which gives rise to exaggeration of reflex activity and to the frequency of twitchings and jerkings, the semeiological value of which is thereby greatly diminished.

In this respect they may be considered as transitional phenomena with regard to insomnia, of which they represent the slighter forms, proceeding from the same causes.

Dreams.—Between certain dreams or visions, which may be considered normal, and nightmare, the pathological nature of which is sufficiently indicated by the name "nocturnal asthma," which is quite as frequently applied to it, the limit is so slightly defined—at least from the point of view of their symptomalogical value—that we can study both under the name of "morbid dreams."

This point of pathology has not yet been examined as it ought to be. However, it should be noticed that the majority of the maladies have a slow onset, and their symptoms remain for a long time unnoticed. Now it is possible that though unnoticed in the waking state, the pathological disturbance becomes sensible during sleep, and thus produces dreams to which their relations with the affected organ give a real semeiological value. On the other hand, dreams assume very special characteristics in certain neuroses (hysteria), intoxications (alcoholism); in fact, in those already predisposed they may have an influence upon the form of the nervous affections (hysterical paralysis, hallucinations, forms of mental aberration).

In the disturbances of the *circulatory apparatus* the dreams are frequently very short, and terminated abruptly by waking with a start. They bear upon tragic events, are accompanied by anxiety, and fears of death are prominent.

Affections of the *respiratory organs* produce horrible dreams. They are those in which one notices nightmare in the pathological sense of the term. The dream is often about animals, which the dreamer imagines he sees seated on his chest, or fastened to his throat, followed by extremely painful sensations of suffocation and anguish. Certain kinds of dreams also are more or less usual in persons the subjects of disturbances of the digestive organs, more especially of the stomach. According to several authors, the nocturnal terrors of children point to intestinal indigestion. Dreams associated with disorders of digestion are usually connected with gustatory hallucinations accompanied by sensations of weight in the epigastrium.

But it is most especially in *nervous affections* that dreaming acquires a semeiological importance. The repetition for some time prolonged, of the same nightmare, is by itself equivalent to what is called "signs of degeneration." There are cases of this kind in which a nightmare instals itself, so to speak, permanently, and for years regularly torments the person. On the other hand, in certain morbid conditions such as hysteria, one notices a constant habit of terrifying dreams with visions of animals, just like those of alcoholism. In these cases the dreams may be followed by paralysis or abasia. It is also to be noticed that in cases of paraplegia of a similar nature it is unusual for the patients to dream of walking.

Finally, it is necessary to mention the dreams in hysterotraumatism, which are almost always said to give rise to the neurosis and have such constancy that they should be enumerated amongst the symptoms. Let it be added, that mental aberration and general paralysis may be ushered in by strange dreams curious in relation with the ulterior hallucinations, and that the dream, in this respect, would have a certain influence over the development of the delirium.

As to *alcoholic intoxication*, it is known that it produces among other symptoms frightful dreams of animals, rats, mice, &c., at other times the dreams relate to the occupation of the patient. However that may be, the peculiarities of the dream have sufficient importance in the diagnosis of alcoholism to prevent negligence in seeking for them.

II.

We must classify as *pathological sleep* a certain number of states more or less analogous to sleep but very different from natural sleep, and it is very important to know how to differentiate the one from the other. They are, hypnotism, the vertigo of paralysis, narcolepsy, hysterical sleep, hypnotic sleep and somnambulism.

The *sleeping sickness* (MacCarthy), or "sleeping dropsy" of negroes, is prevalent on the western coast of Africa.

Three degrees can be distinguished between the tendency to drowsiness and complete lethargy. The first symptom of the disease consists in the eyelids being half closed at the same time the person is taken with a fit of drowsiness, at different times of the day; then these fits become almost continuous, the patient with difficulty aroused to eat, and falls asleep again almost immediately. Finally, the sleep becomes persistent, and different excitations no longer have power to arouse the patient, who thus succumbs after a sleep lasting for three or four days, presenting in certain cases a notable diminution of temperature. This affection is unaccompanied by motor or sensory disturbances, and has a mean duration of three or four months. The characters of this condition are so pronounced, and its geographical distribution even so exact, that it is unnecessary to discuss its differential diagnosis.

Paralysing vertigo has been observed endemically in Switzerland, and appears to be a modified form of the preceding affection.

This affection is characterised by headache, vertigo, enfeeblement of the muscular system, with staggering gait and double ptosis. The disease lasts some months, and always terminates favourably. In relation to this, it is convenient to mention another variety, described by Garget, occurring in a person who, after an explosion, was seized with violent excitement accompanied by diplopia and visual troubles. Following on this, double occulo-motor paralysis with ptosis, progressive muscular weakness with intellectual apathy and tendency to sleep. There were no paralytic or sensory troubles. The patient after some relapses and remissions slept for five months and then died. This affection seems to be related to acute poliencephalitis (*Mauthner*) ; it is comparatively rare, but it is, nevertheless, necessary to be warned of its existence in order to avoid confusion with the following forms.

Narcolepsy would not constitute a pathological entity but only a symptom (unlike insomnia), possibly appearing in widely divergent cases. It consists essentially in a *pathological exaggeration* of the desire to sleep. The tendency to sleep is constantly seen in the youth of both sexes; it is irresistible, comes on for the most part suddenly, it recurs sometimes at very short intervals, and with sufficient strength to cause the patient to yield inevitably as soon as it is felt (Ballet). The tendency to sleep is sometimes spontaneous, at others it is brought about by the most diverse external influences, movements or emotions. It is more or less intense as the case may be, varying from an apathetic condition to deep sleep. This sleep has in other respects a normal appearance. The patient does not dream, and he can only be awakened by the most energetic excitations.

The condition of narcolepsy has been met with in a certain number of cases in which it was impossible to make out any other morbid alteration ; most frequently it is a question of cardiac affection, of dilatation of the stomach, of general obesity, of diabetes, or of hereditary neuropathy.

The diagnosis of narcolepsy would in itself be easy, but it is sometimes more difficult to make out the morbid condition giving rise to it. For this purpose one should not neglect to make a careful examination of the heart, digestive, functions, and more especially of the urine, for this has proved the presence of diabetes which up to that time had been unrecognised. We consider that hysterical sleep, as well as the different varieties of hysterical somnambulism which some authors have classed as narcolepsy, has sufficiently pronounced symptoms of its own to merit a special description. Indeed, we place *hysterical apoplexy* along with *hysterical sleep*, as it is impossible to differentiate them nosographically, according to the teaching of M. Charcot, for it is a question of one and the same morbid condition; the only difference being that in a case described under the name of hysterical apoplexy (Debove and Achard), the sleep attack has a sudden onset and is followed by hemiplegia, while this paralysis may be absent in other cases.

In a great number of observations the attack of sleep is preceded or followed by usual phenomena or by premonitory indications of a convulsive attack and habitual hysteria. At other times the onset is sudden, and analogous to that of apoplexy due to cerebral hæmorrhage, and one may see patients who are attacked by it fall suddenly, it may be in the midst of a conversation, or in the midst of their occupation.

Again, the sleep crisis may come on after the attack. The sleep having once come on, the patient usually has a placid expression, but sometimes the features are a little drawn; the respiration is not stertorous, and the patient does not snore. The limbs may be quite limp, or, perhaps, more frequently in a state of general rigidity; it may attack the lower extremities only, or even a single limb.

One constantly notices a kind of vibration of the eyelids, the more so on attempting to open the eyes, which converge upwards and inwards. Trismus exists in almost all cases (Gilles de la Tourette). Respiration is regular, the pulse quiet, the temperature may rise a degree and some tenths (38.9), but never more. The urine is passed in the bed, and it has a chemical composition characteristic of the convulsive attack; it is diminished in quantity and in its constituents, and there is an inversion of the formula for the phosphates (Gilles de la Tourette and Cathelineau).[1] The sleep is also characterised by this fact, namely, the strongest excitations—noises, inhalations of irritating vapours, faradisation of the skin, nerve trunks and of the muscles—produce no reaction. It is equally noticeable that during the sleep one often sees the patient execute certain movements, as opisthotonos, movements of salutation, &c., at regular intervals without awaking.

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[1] These authors have observed that the relative proportion of the earthy phosphates to the alkaline phosphates contained in *normal urine* is 1 to 3; but in *hysterical urine during the attack* the proportion is equal. It is this alteration that they refer to by the term "inversion of the formula of the phosphates."

Lastly, there sometimes exist hyperæsthetic points which are at the same time hysteria-producing zones, pressure upon which brings on an attack which puts an end to sleep.

This condition should be differentiated from natural sleep, from *coma*, and from the other pathological conditions of sleep previously mentioned.

Natural sleep terminates under the influence of various excitations of the senses—a fact which is sufficient to distinguish it. The knowledge of the antecedents and of the commencement of the attack, if it was ushered in by an hysterical crisis, would usually be sufficient to establish the diagnosis; but in the absence of these points hesitation would be possible. One would then have to rely on the general aspect of the patient, on the temperature, on the presence of rigidity (trismus, contracture, palpitation of the eyelids) and on the alterations of the sensibility, in order to diagnose it from coma.

In *narcolepsy* the sleep is neither accompanied nor followed by hysterical phenomena; it presents none of the preceding characteristics. In the following paragraph we shall see in what way it is distinguished from hypnotic sleep.

Hypnotic sleep presents very various phenomena, according as it is observed in different individuals, and, moreover, it assumes very distinct forms. It can be divided at the outset into *grand* hypnotism, *petit* hypnotism.

The "*grand hypnotisme*" (Charcot) occurs in hysterical persons. It comprises three periods or phases, each possessing somatic characters peculiar to itself; (*a*) the lethargic state; (*b*) the cataleptic state; (*c*) the somnambulic state.

We shall only describe here the two first states, the third being more appropriately considered in the paragraph below, under the heading of *somnambulism*. The commencement of the lethargic period of the *grand* hypnotism is frequently marked by a deep inspiration, accompanied by a slight laryngeal bruit, and it is not uncommon, at the same time, for a slight froth to appear on the lips. The eyes are closed, the eyelids vibrate slightly; the head, the trunk, and the whole muscular system are in a state of complete relaxation; anæsthesia is complete. The characteristic sign of this state is the property which the muscles possess of contracting under the influence of mechanical stimuli, applied either to the muscles themselves or to the nerves distributed to them (the neuro-muscular hyper-excitability of Charcot). If a certain amount of pressure be applied to the belly of a muscle, it soon contracts, and this contraction only disappears on working up the antagonistic muscles. Again, pressure applied to the trunk of a nerve produces immediate contraction of all the muscles supplied by that nerve, and this contraction yields, like the preceding one, under the same influence.

These symptoms are more or less marked according to the individual, and they are sufficient to affirm the diagnosis. However, this same tendency to contraction may exist in hysterical sleep, but then, if one were so enlightened as to the facts, one would diagnose the lethargic state by proving that a slight breath of air on the face quickly dissipates it. The cataleptic stage of the *grand* hypnotism is characterised by the complete immobility of the subject, and, at the same time, by the peculiarity that the limbs remain in any position in which they may be placed. The eyes are open, the eyelids do not blink, tears run down the cheeks, the gaze is fixed, and the face is immovable. Anæsthesia is complete, as in the preceding state. If an arm be raised, or if the body be placed in any attitude, whatever the position thus assumed is maintained for a considerable time without effort. Moreover, there exists a kind of suggestion by the muscular sense—that is to say, that the attitudes in which the limbs are placed give rise to corresponding expressions on the face. If, for example, the hands of the subject are placed in an attitude of prayer, the countenance becomes ecstatic.

Hypnotic catalepsy is comparatively easy to diagnose from these signs. Simulation can be detected by proving the fact that in true catelepsy the limbs retain the position in which they are placed without any sign of effort on the part of the patient, as acceleration of the respiratory movements. In *hysterical sleep* one sometimes observes the same peculiarity of the limbs to retain their position, but then these positions do not cause corresponding expressions of the countenance as in hypnotic catalepsy. In cases of *catatonia* sufficient indications are given by the facts, and the mental condition of the subject.

The "petit hypnotisme" has various degrees—drowsiness, light sleep, deep sleep, very deep sleep, somnambulism (Bernheim). In the first degree it is only a question of heaviness and numbness; in light sleep, the persons sleep but hear any conversation that is taking place; in deep sleep they lose all remembrance of what has happened during sleep, but they are still in relation with those who are present as well as with the operator. In very deep sleep the *isolation* of the person is complete, and he is in relation with the operator only.

In addition to these signs there are also present anæsthesia and more especially a marked docility to the suggestions of the operator. This last characteristic would be sufficient to establish the diagnosis of hypnotism; but in all cases of the *petit* hypnotism, in the absence of objective symptoms, one must rely on the good faith of the patient.

Somnambulism comprises a certain number of conditions, all characterised by an automatic activity of the nerve centres, which causes those who are the subjects of it to perform unconsciously a variety of different actions.

We shall adopt for the description the following classification (Charcot's) of somnambulisms:

Physiological				Natural S.
Pathological	Epileptic	Minor Automatism		Epileptic S.
		Major Automatism		
	Hysteric	Spontaneous	Prolonged, isolated, transformed, passional crises	Hysteric S.
		Artificial		Hypnotic S.

Natural or physiological somnambulism has long been recognised. The somnambulist of this variety, according to the etymology of the word, is "one who walks during sleep."

It has been defined thus: "Somnambulism is when the functions performed during the period of wakefulness are repeated during sleep, which, in other respects, is natural"

It is seen in children and usually occurs in the middle of the night. The person goes to bed as usual and after some hours' sleep, suddenly, or after a slight momentary restlessness, gets up; then during a longer or shorter period he performs very various actions, goes back again to bed, and on awaking has no recollection of what has taken place during the night.

The somnambulist has his eyes wide open, but keeps them fixed, the pupils are contracted and he guides himself without hesitation, apparently only seeing the objects or persons which are playing a part in the episode he is enacting. Some somnambulists appear to hear; this fact, however, is less well-established; when spoken to in a loud voice they generally take no heed, and continue the actions they have begun.

The muscular sense is remarkably developed in them, and it is to the hyperæsthetic condition of this sense that the facility with which they often extricate themselves from perilous positions, as jumping, balancing themselves on the roof, &c., is to be attributed. Moreover, the tendency to spasmodic muscular contractions, which mark the hysterical somnambulist, does not occur in these cases.

On awaking—and this is a very important point—the somnambulist has completely forgotten all that he has done during the attack. He performs very different actions, but speaking in a general way, he always appears to be acting a dream, which comprises one or more of a series of quite distinct episodes.

From these signs it is comparatively easy to distinguish natural somnambulism, if one takes into account the entire normal condition of the patient in the interval between, and the nocturnal character of, the attacks. These characters differ from those of *epileptic* somnambulism. Every form of epilepsy comprises a species of automatism, usually rudimentary, which may, under certain conditions, acquire considerable development.

The simple and most common forms are those which partake of the nature of the *petit mal*, the *petit automatisme*. Sometimes the trouble in these cases lasts only a few moments, and then the patient continues, for instance, the work with which he was occupied at the onset of the attack. At other times the fit is of longer duration and more complex. In a case given by Trousseau, a magistrate sitting at a learned society suddenly goes out of the hall without his hat, walks a hundred yards outside, then returning takes part in the discussion without having any recollection of what he had done.

Cases of this description are comparatively easy to diagnose, for the psychical and impulsive characters of these attacks are seldom the only ones which mark the subject of them. Most frequently he has true vertigo, or even convulsive epileptic seizures, and, in a certain number of these cases, these signs immediately precede the psychical disturbance.

The difficulty is much increased when other forms are in question, *epileptic ambulatory automatism* (Charcot). The attack may last from two hours to six days, during which the patient performs a series of capricious movements with a desire to walk about; he is quite unconscious, and has the appearance of an ordinary man, but retains no recollection of his movements. The diagnosis would be easy should the patient have any history of antecedent convulsive crises, and above all if the onset of the period of unconsciousness is marked by an aura.

But it may not be so very easy. The diagnosis is then based rather on the negative symptoms—absence of hysterical signs and crises—and on the analogies presented by these unconscious impulses, and those which follow the fit of sleep; and lastly, it is by noting most carefully the positive results of treatment with bromide, that we should be justified in designating these cases as epileptic somnambulism. *Hysterical somnambulism* is marked by attacks which are usually preceded or followed by the motor phenomena of a hysterical fit, but more rarely these are manifested some time previously.

Hysterical somnambulisms may be considered as transformations of the phase of the writhing attitudes of the hystero-epileptic attack. This phase is characterised by delirium, in which the patient, by his words and gestures, appears to have hallucinations of various descriptions—cheerful, sad, mournful, terrifying, &c. The animated performances in which these persons are then taking part usually commence in the same order and proceed in a kind of series.

In the first group of acts there is an hysterical attack in which the convulsive period is shortened and the emotional phase is lengthened and simulates somnambulism. When the attack is coming on it begins with some epileptiform movements of short duration; then the phase of delirium supervenes, bringing with it, as usual, a succession of scenes always the same. It is then that the patient is a true somnambulist; his eyes are open, at least most usually, but he only sees the dream which he is following out—he walks, gesticulates, talks, cries or sings. Sometimes a word or certain noises may occur during the delirium, but they only serve to modify the order of succession of the phases; no new one is added, that is to say, no suggestion has any creative influence. Finally, it is often possible to bring on an attack by hypnotism, or, on the other hand, it can be arrested by pressure upon the hystero-inhibiting zones.

In another variety of seizure there is no abnormally prolonged phase of the attack, but a completely isolated phase. The patient enters upon the scene which he is about to enact by some twisting movements of the hands, which represent the shortened convulsive period of the attack; he then proceeds to the performance of certain actions in accordance with the hallucinations from which he is suffering.

In this case external intervention has now more effect not only in modifying the scenes of the delirium, without, however, adding anything new to it, but it elicits answers from the patient to questions put to him. Otherwise the patient is not amenable to suggestion, and shows none of the contracture of somnambulism; but his attitude is more quiet, his activity diminished, his passivity increased.

And finally, in the last class are the cases usually considered as examples of "double identity." These are cases of people living, so to speak, a two-fold existence; that is to say, passing alternately from one state to another, so that everything that happens in state No. 1 is only known in a similar state, and the same thing occurs in state No. 2.

The subject in reality exists as two personalities, each unknown to the other and differing in psychical and sometimes even in physical characteristics. In fact, there may exist in one of these states hysterical symptoms (hemianæsthesia, contracture, &c.), which may disappear in the other condition.

It is evident that all cases of this kind have characters so prominent, that there is no need to dwell more upon the diagnosis of them.

Hypnotic somnambulism is the third stage of the *grand* hypnotism (Charcot), or the fifth degree of the *petit* hypnotism (Bernheim).

This state is marked by less spontaneous activity on the part of the subject, and his passive obedience to every suggestion which it may please the experimenter to make to him. There is also present a marked exaltation of the muscular power, and, above all, of the special senses.

The general sensibility is usually diminished, or entirely absent. The eyes are wide open, and the subject appears to be awake, but in the *grand* hypnotism there is a physical sign which allows us to ascertain the reality of this state. This sign consists in a muscular hyper-excitability, which may be produced by a superficial excitation of the skin. A breath, or the slightest tickling, causes contracture, which disappears by manipulation exercised on the antagonising muscles. On awaking, the somnambulist has forgotten all that has passed, but he may retain the recollection during another period of somnambulism. Lastly, there remain a large number of conditions called *traumatic amnesia*, which have certain points of resemblance with the automatic states which we have just described, but their nosographical history is not yet sufficiently made out to warrant their description here. Nevertheless, their eventuality is foreseen, and ideas as to their causation would then establish the diagnosis.

Reviews and Notices of Books.

Jean Jacques Rousseau Krankheitsgeschichte. By P. T. Möbius. Gr. in 8, pp 192. Leipsig: Vogel, 1889.

La folie de J. J. Rousseau. Par le Dr. Chatelain, ancien médecin en chef de la maison de santé de Préfargier. Neuchatel: Attinger frères, 1890.

The mental state of J. J. Rousseau has from the very outset been a subject of discussion among literary men. In the course of last century, whilst the great Genevese philosopher was still alive, it became the fashion among his detractors to call him a lunatic. After Voltaire and the Encyclopædists, such terms of opprobrium were still bandied about by the opponents of his philosophy; but these expressions, being inspired merely by hatred and prejudice, were devoid of any scientific weight, and denoted moral short-comings among those who used them rather than proved mental deficiency on the part of the subject of their vituperation. The supporters of Rousseau were fully conscious of this fact, and passionately defended their hero against all such imputations.

It is, however, impossible for us now to dispute the fact that Rousseau, during the latter years of his life, became the victim of a true progressive affection of his intellect. The evidence adduced by Drs. Möbius and Chatelain is absolutely conclusive.

The scientific investigation and impartial criticism of these two distinguished physicians throw a clear light upon the actual nature of Rousseau's mental aberration. His insanity did not bear upon his genius and brilliant perceptions. His *Confessions*, *Dialogues*, and *Promenades d'un Solitaire*, which were written during the culminating period of the malady, are literary masterpieces intended to defend his memory from the attacks of persecutors by whom he felt himself surrounded. We fully agree with Möbius when he describes Rousseau as a paranoiac, with symptoms of Kraepelin's "combining persecutional delirium."

Chatelain, who also places Rousseau among the "persecuted" insane, had already mentioned his case in a previous volume

(*La Folie*, Neuchatel, 1889) under the heading of systematised delirium. This author has had access to documents unknown to Möbius, such as a memoir by Prof. E. Ritter, of Geneva, and the researches of the archivist, Dufour-Vernes; these materials, however, do not throw much light upon the question of an hereditary taint among Rousseau's ancestors. On the other hand they supply interesting details concerning the life of these people, and reveal peculiarities of character and behaviour which have exercised an obnoxious influence upon their illustrious descendant.

I have also investigated this subject in the Archives of Geneva, and can testify that up to the fifth generation of Rousseau's ancestors no case of lunacy, suicide, or nervous affection are mentioned in the registry of deaths; hence we cannot agree with those who, like Möbius, admit as uncontrovertible the existence of hereditary tendencies ("on the paternal side") in the case of Rousseau; all evidences, on the contrary, lead us to consider the mental perturbation of his latter years as the result of untoward circumstances, such as a lonely forsaken childhood and a restless distracted existence.

The instability of temper in the case of Isaac Rousseau, the father, and the flight of the elder brother do not amount to proof of a neuropathic taint; and the story related by Corancey and Musset-Pathay, of a cousin born in Persia and suddenly losing his reason in the forest of Fontainebleau, is too apocryphal to carry any weight. Moreover if it were true, nothing shows, as Möbius himself admits, that the "Persian cousin" did not succumb to a mere attack of alcoholism.

Did Rousseau present any symptoms of degenerescence? There is no evidence entitling us to answer this question affirmatively. Möbius discusses from this point of view Rousseau's congenital malformation of the bladder, which troubled him so much throughout the period of his manhood. But this was the only physical defect he presented, and we cannot agree with Chatelain's view of the case. It is well known that the Frère Côme had diagnosed a hypertrophy of the prostate, which was not found at the autopsy. Möbius is probably right when he thinks that Rousseau's was a case of the vesical anomaly described by Mercier, who, like Lallemand, regarded it as the cause of all the patient's hypochondriac symptoms. However exaggerated this extreme view may be, it may be safely assumed that Rousseau's lifelong moral depression was connected with his local trouble.

The philosopher's mental life may be divided into several periods, each presenting a special group of symptoms. Three such distinct periods may be distinguished. During his youth he suffered from neurasthenia, of which he himself gives a life-like description in Books V. and VI. of his *Confessions ;* later on he becomes the vesical hypochondriac just alluded to ; finally, during the last twelve years of his life, he showed symptoms of persecutional delirium.

Both Möbius and Chatelain declare that this delirium began when Rousseau, aged fifty-four, was in England. Möbius points, as containing the first indications, to a letter written from Wootton (Derbyshire) on July 10th, 1766, in order to substantiate his accusations against Hume. This letter indeed contains characteristic passages, and from this moment the delirium more and more clearly shows periods of exacerbation, followed by remissions every time the patient moves into another locality. Was Rousseau the victim of hallucinations ? Though Chatelain seems disposed to admit it, no such evidence has been adduced as to prove that this has been the case. Möbius seems to hold the correcter view that though the sufferer may have been misled into drawing erroneous inferences, he never became the victim of sensory delusions, nor hallucinations.

Both authors make a thorough study of the progress and development of Rousseau's mental disorder, though from two different points of view. Möbius shows us the patient during the several stages of exacerbation and remission, which coincide with his stays at Trye, Bourgoin, Monguin and Paris. Chatelain endeavours to depict the general characteristics of the delirium, which he studies in a general manner, in its causes as well as in the actions and writings of the patient. Möbius brings forward the four letters to M. de Malesherbes, which well display Rousseau's temper and explain his motives and behaviour ; then consecrates a whole chapter to the "Dialogues," in which are found the documents shedding most light upon his delirium. This is what Chatelain says of these Dialogues :—

"Here Jean-Jacques places himself in the foreground ; and this work marks the climax of his symptoms, which it embodies not only in matter, but in form . . . Everywhere one becomes conscious of the obsession and the painful effort of the lunatic seeking, with sweat on his brow, the exact expression embodying his anguish, fearing to write inaccurately or insufficiently, losing himself in details, ever pacing round the same circle of ideas like a wild beast in its cage."

Möbius remarks that persecutional delirium usually bursts forth in youth : hence Rousseau's predisposition to it cannot have been pronounced since he was attacked at so late an age as fifty-four; and this is another argument against the hereditary neuropathic taint of which we have already spoken. Möbius says: "Rousseau's goodness of heart and power of intellect appears in the midst of his disease. His moral character and his tender nature saved him throughout from any extravagant action, or excess of word or deed against his imaginary persecutors."

After a period of severe struggles a slowly progressive mental decay became evident, lasting till death—not destructive enough, however, to prevent Rousseau from active work, as is evidenced by his last writings, the *Rêveries d'un Promeneur solitaire,* full of resigned tranquillity.

The false report of Rousseau's suicide, so widely spread about, is, we are glad to find, rejected by both our authors. It is easy indeed to convince oneself, by an impartial sifting of all the trustworthy evidence we have concerning the circumstances of his death, that Rousseau did not destroy himself, as some writers suggested notwithstanding the energetic assertions to the contrary from those who alone were in a position to know the facts.

To those interested in the captivating problem of "genius and madness" we cannot too warmly recommend the volumes just summarily reviewed.

A. LADAME, M.D.

Railway Injuries in their Medico-legal and Clinical Aspects.

HERBERT W. PAGE, M.A., M.C.Cantab., F.R.C.S.

MR. PAGE has undoubtedly added a valuable contribution to the literature of a chapter of medicine and surgery on which a strong light is needed. His long experience as surgeon to one of the large railway companies gives any opinion he may express on the subject great weight. The reader will feel that no undue amount of sympathy is given to the real sufferer, while the impostor will be relentlessly exposed. This somewhat judicial attitude is, we suppose, quite unavoidable, and in our opinion rather adds to the value of Mr. Page's work. On the other hand, however, we cannot help thinking that in his comments on many of the admittedly genuine cases, we detect something of the spirit of counsel for the company.

The first chapter calls for little comment, being devoted to the injuries of the back. These injuries are believed to be of the nature of sprains to ligaments and muscles, the predominant symptom being pain generally out of proportion to the degree of ascertainable injury.

The next two chapters treat of general "nervous shock," or the condition known as neurasthenia. The picture of this class of patient is well drawn, and illustrated by not too many well-selected cases. An attempt is made to explain the curious and often anomalous symptoms of the neurasthenia, by supposing him to be morbidly alive to the "perception of the sensations of organic life" (p. 52). Under normal circumstances, such sensations if felt are disregarded, but if some sudden shock be experienced "the intellectual control is lessened, while the organic sensations declare their being and force themselves into the conscious life of the individual."

We agree with Mr. Page that the effects of a collision are often less physical than psychical. We can therefore perfectly understand how profoundly litigation must affect the progress of the case, so much so as to give rise to a class of symptoms which he calls "litigation symptoms." We fully sympathise with Mr. Page in the difficulty he experiences in distinguishing real disability from, let us say, unintentional exaggeration. In ordinary hospital practice, in cases in which the question of legal damages does not enter at all, we find this same tendency to exaggerate, and so frequently in *bonâ-fide* cases, that it may be said to be part of the affection. It is obvious from this how great an opportunity for imposture is offered to unscrupulous persons, and how extremely delicate is the position of the medical man in such cases.

In the fourth and fifth chapters we have a discussion of the functional disorders following injury. At the outset we are confronted with the difficulties in nomenclature. Mr. Page uses the term "Fight neurosis," as involving less than the more usual "traumatic hysteria." A very obvious objection to the name hysteria, is its association in the public mind with "shamming," by which serious injustice may often be done to a patient. The term "functional," is perhaps least objectionable, as a provisional term, to include disorders which have not at present been found to have any *visible* pathological basis. We agree with Mr. Page (p. 62) that there is a tendency to assume the absence of such a basis, because it has not been found.

The most striking functional manifestations with which Mr. Page appears to be familiar as sequelæ of railway injuries, are acute hysteria, sobbing, laughter, &c., soon after the accident, paralyses, such as hemi paresis, paraplegia, contractures, disorders of sensation, anæsthesia, analgesia, and convulsive seizures. The question of hemi anasthesia is discussed at some length, but we note the absence of any reference to contraction of the field of vision and achromatopsia which so often occur with it. In the diagnosis of affections in which we have to depend so much on the statement of the patient, a sign such as this is of peculiar value, and if found to co-exist with anæsthesia must be regarded as confirmatory of the genuineness of the latter.

The greater part of chapter four is given up to the discussion of the causation of functional affections. That they are of cerebral origin we are inclined to agree with Mr. Page, but we cannot at the present stage of thought on the subject go any farther with him. He institutes a parallel between the mental state of the patient after shock, and the subject of hypnotism. Having admitted the existence of such a condition, the next step is easy, namely, that the functional manifestations are due to "suggestion." Such "suggestion" is supplied by the nature of the injury, or by the fears and expectations of the patient himself, and to it he gives the name "auto-suggestion." He even hints at the possibility of the medical examination acting as a "suggestion," for instance, of the extent of anæsthesia (p. 67). We fail to see how this could apply to so complicated an examination as the drawing of a perimeter chart. The theory might account for some of the grosser forms of hysteria, but in our opinion, not for the final functional manifestations. For instance, we submit that the fact of the convulsive seizures being sometimes initially under the control of the will (p. 73), and we believe such cases to be comparatively uncommon, is of little importance in this connection, because there is no doubt that the same may be said of true epilepsy. The so-called hystero-epileptic fit is in the majority of cases as far out of the control of the patient as is the ordinary true epileptic fit. If the fit were a product of "suggestion" why should it differ so essentially as it does from the epileptic discharge, and why should it offer characteristics so strikingly *sui generis*, that the trained observer can recognise it almost at once. We contend that the typical functional fit is as much a distinct disease or sign of disease as an epileptic fit. What the underlying morbid condition is, we must confess at present profound ignorance.

Chapter six contains useful hints on treatment.

Chapter seven is on malingering, without which, unfortunately, a work on railway injuries could scarcely be said to be complete. The last chapter is medico-legal. The book is very clearly written and in a scholarly style; the division of the chapters into paragraphs with marginal headings is of the greatest use to the reader.

TOPOGRAPHIE CRANIO-CEREBRALE. *Applications Chirurgicales.* By Le Fort. *Paris:* 1891, pp. 160.

WE are sorry that this work, which has evidently been designed with much care, and which includes all the important literature of the subject, has not been made more complete by the addition of various observations and material, more especially those accumulated by Cunningham, for we think then that very grave inaccuracies such as are freely exhibited in some of the drawings (notably Pl. II.) would have been avoided; but while we feel obliged to make this unfavourable criticism to commence with, we must congratulate the author on the very thorough way in which he has stated the localisation of the chief sulci and convolutions as determined by all the other authors who have written on the subject. In this way the work is an extremely valuable summary. In collaboration with M. Debierre he has introduced what he calls an "auto-gravure" method of determining the situation of the sulci. This is a modification of that published by Messrs. Anderson and Makins; but it has the misfortune of necessitating the exposure of a certain area of the dura mater, which is then opened and lines drawn on it within, opposite the sulci. After the lines are thus traced on the skull they are checked off by driving pegs through the bone. This method unquestionably gives a rapid and ready means of localising the convolutions, and has considerably added to our knowledge of the subject, but it obviously is not free from error by deformation. In addition, M. Le Fort gives a valuable series of observations of the variations in the topographical relations according as they are produced by age, and he discusses those less known which are produced by race. Finally, he summarises in tabular form the proportionate distances according to the proportionate diameters of the skull, and further supplies a literature of the subject which will be of much interest to those who are desirous of obtaining information on this question.

VICTOR HORSLEY.

Abstracts of British and Foreign Journals.

Current Nerve Anatomy and Physiology. By ALEX—HILL, M.D., Master of Downing College, Cambridge.[1]

ANATOMY.

1. Anatomy of the Central Nervous System and of the Spinal Cord in particular. SINGER AND MÜNZER (*Denkschr. der Math.-Naturw. Classe der K. Akad in Wien*, lvii., pp. 21, pls. i., ii. and iii., *Sep. Abd. Wien*, 1890, *Tempsky*).

SURFACE ANATOMY.

2. Some Points in the Surface Anatomy of the Brain. BENEDIKT (*J. of Anat. and Phys.*, xxv., 2, pp. 210-221, Jan., 1891).

TOPOGRAPHY.

3. Cranio-Cerebral Topography. DANA (*J. of Nervous and Mental Diseases*, xvii., p. 407, 1890).

COMPARATIVE ANATOMY.

4. The Convolutions of the Brain: a Study in Comparative Anatomy. TURNER (*Williams and Norgate, London*, 1890, pp. 53, figs. 42).
5. The Brain in Edentata. RABL-RÜCKHARD (*Arch. f. Mikrosk. Anat.*, xxxv., 2, p. 165, 1890).
6. Brain of the Seal. SPITZKA (*Am. Naturalist*, xxix., p. 115, 1890).
7. Dr. Theodor's Monograph on the Seal's Brain. SPITZKA (*Anat. Anz.*, v., 6, p. 173, 1890).
8. Brain of the Alligator. HERRICK (*J. Cincinnati Soc. Nat. Hist.*, 1890).
9. The Brain of the Lizard. KÖPPEN (*Wien Klin. Wochenschr.*, xiii., p. 255, 1890).
10. The Structure of the Brain in Myriapods and Arachnids. SAINT REMY (*Rev. Biol. du Nord de la France, Lille*, 1889-90, ii., p. 41).

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[1] In completing the list of titles of papers published in 1890, extensive use has been made of the synopses given in the *Centralblatt für Physiologie*. It is hoped that the translation of all foreign titles into English will facilitate the search for the literature of any given subject.

11. The Minute Anatomy of the Fore-brain of Amphibia. OYARZUN (*Arch. f. Mikrosk. Anat.*, xxxv., p. 380, 1890).

MORPHOLOGY.

12. Origin of the Nerve-centres in Coelomata. ROULE (*Arch. de Zool. Exp.*, viii., 1, p. 83, 1890).
13. Origin of the Cerebral Cortex and the Homologies of the Optic Lobe-layers in the lower Vertebrates. NAKAGAWA (*J. of Morphology*, iv. 1, p. 1., 1890).

EMBRYOLOGY.

14. The Development of the Vagus, Accessory, Hypoglossal and first Cervical Nerves in Sauropsids and Mammals. CHIARUGI (*Archiv. ital. de Biol.*, xiii., 2, 1890).
15. Some Stages in the Development of the Brain of Clupea harengus. HOLT (*Proc. Roy. Soc.*, xlvii., p. 199, 1890).
16. The Development of Peripheral Nerves in new-born Children. SOKOLOW (*Wratsch*, Ni. 17 and 20, 1890).

PHYSIOLOGY.

17. The Brain, its Functions, Practical Applications. WARNOT (*Brussels*, 1890).
18. The Functions of the Brain. Doctrines of the Italian School. SOURY (*Arch. de Neurol.*, xix., 55 and 56, 1890).
19. Notes on the Functions of the Nervous System. GOWERS (*Lancet*, 1890, pp. 955, 1006, 1113, 1116).
20. The Co-operation of Parts of the Brain. MEYNERT (*Berlin*, 1890, *Hirschwald*).

PSYCHO-PHYSIOLOGY.

21. Psychology in relation to the latest views as to the Physiology of the Brain. BIANCHI (*Milan*, 1891, *Rechiedei*, pp. 17).
22. Sympathizings (mitempfindungen) and related Processes. QUINCKE (*Zeitschr. f. Klin. Med.*, xvii., 5, 1890).
23. The Concurrence of Psychological States. BINET (*Rev. Philosoph.*, xv., p. 138, 1890).

CEREBRUM: ANATOMY.

24. The Frontal Brain. EBERSTALLER (*Urban und Schwarzenberg, Vienna and Leipsic*, 1890).

CEREBRUM: COMPARATIVE.

25. The Comparative Anatomy of the Frontal Lobe. ZUCKERKANDL (*Mitth. d. Anthrop. Gesellsch. in Wien*, xix., 6, 1890).

CEREBRUM: HISTOLOGY.

26. The Minute Anatomy of the Forebrain in Amphibia. OYARZUN (*Arch. f. Mikrosk. Anat.*, xxxv., p. 380, 1890).

CEREBRUM: PHYSIOLOGY.

27. Demonstration of a Dog, in which the Cerebrum is Extirpated. GAULE (*Correspondenzbl. f. Schweizer Aerzte*, xx., 10, p. 319, 1890).
28. The Physiology of the posterior part of the Great-brain. KORÀNYI (*Centralbl. f. d. Med. Wissensch.*, Ni. 28 and 29, 1890).
29. Experimental Stimulation of the Brain in Man. SCIAMANNA (*Arch. di Psichiatria*, x., 5, p. 497, 1890).

CORTEX: ANATOMY.

30. Bearing of Secondary Fissures upon the Explanation of Brain-fissuring. FLESCH (*Anat. Anz.*, v., 16 and 17, p. 494, 1890).
31. The Sylvian Fissure and the Island of Reil in the Primate Brain. CUNNINGHAM (*J. of Anat and Phys.*, xxv., 2, pp. 286-292, Jan., 1891).

CORTEX: PHYSIOLOGY.

32. Movement-and-Sense-pictures of Man in their relation to the Cerebral Cortex. CARO (*Hamburg*, 1890).
33. Rôle of the Motor Convolutions in the New-born. HERZEN (*C. R. du Congrès intern. de Psychologie Physiol., Paris*, 1890, p. 125).
34. The Physiology of the Cerebral Cortex. MARXOW (*Centralbl. f. Physiol.*, iv., 18, p. 537, Dec. 6, 1890).
35. The Investigations of the Functions of the Cortex of the Brain. UNVERRICHT (*St. Petersburg Medical Journal*, 1890, No. 21, p. 192).
36. The Function of the Cerebral Cortex. MUNK (*Collected Works, Hirschwald, Berlin*, 1890).
37. The Cerebral Cortex and the central origin of Nerves. MARTINOTTI (*Soc. Med.-Chir. di Pavia*, 1889).
38. Bilateral Trophic Troubles after lesion of the Cerebral Cortex. LANGLOIS & RICHET (*C. R. Soc. de Biol.*, May 31, 1890, p. 315).
39. The question of Localization of the Functions of the Great-brain. WUNDT (*Wundt's Phys. Studien*, vi., 1, p. 1, 1890).
40. The Cortical Motor Centres in Man. POSTEMPSKI AND BOCCI (*Bull. della r. Accad. Med. di Roma*, xvi., 4 and 5, p. 176, 1890).

41. Cerebral Localization and Cerebral Epilepsy. Sotis (*Giorn. med. d. r. esercito*, xxxviii., p. 133, *Roma*, 1890).
42. Electrical Excitation of the so-called Motor Cortex and Internal Capsule in an Orang-outang. Beevor and Horsley (*Proc. Roy. Soc.*, xlviii., 293, p. 159, 1890).
43. Numerous Cases of Vivisection practised on the Brain of Man, their Verdict against the Doctrine of Psycho-motor Centres. Brown-Séquard (*Arch. de Physiol.* (5), ii., 4, p. 762, 1890).

Cortex: Sight.

44. Experiments upon the Cortical Sight Centre. Thompson and Brown (*Researches of the Loomis Laboratory, New York*, i., p. 13, 1890).
45. The Visual Area. Bechterew (*Kowalewskij's Arch.*, No. 1, 1890).
46. Cortical Blindness. Förster (*Arch. f. Ophthalmologie*, xxxvi., 1, p. 94, 1890).
47. Double Experimental Blindness in a Dog with Autopsy. Richet (*Rev. philosoph.* xv., 5, p. 554, 1890).
48. Eye-movements on Stimulation of the Visual Area. Obregia (*Du Bois-Reymond's Archiv.*, 3 and 4, p. 260, 1890).
49. Associated Eye-movements produced by Cortical Faradization of the Monkey's Brain. Mott (*Brit. Med. J.*, 1890, p. 1419).

Cortex: Hearing.

50. The Auditory Centre. Ferguson (*The J. of Anat. and Phys.*, xxv., ii., pp. 292-294, Jan., 1891).
51. Auditory Area and Movements of the Ears. Baginsky (*Neur. Centralbl.*, ix., 15, p. 458, 1890).

Cortex: Speech.

52. Facts tending to show that the Right Hemisphere plays a rôle in the Expression of Articulate Language. Luys and Mabille (*Rev. d'Hypnol.*, p. 134, 1890).
53. Localization of the Cortical Speech-centre in Man. Rossbach (*Deutsches Archiv. f. Klin. Med.*, xlvi., 2, p. 140, 1890).
54. The Localization of the Larynx in the Cerebral Cortex. Krause (*Berl. Klin. Wochenschr.*, 25, p. 556, 1890).

Cortex: Trunk-Muscles.

55. Experimental Study of the Cortical Innervation of the Trunk-Muscles. Kusik (*Inaug. Dissertation, Dorpat*, 1890).

CORTEX: VASOMOTOR.

56. Effects of Cortical Stimulation upon the Arteries. SZPANBOCK (*Gazeta Lekarska*, x., 9-11).

CEREBRUM: DUALITY.

57. Double Consciousness in Health. BINET (*Mind*, Jan., 1890, p. 46).
58. The two Brains of Man. DESCOURTIS (*Revue d'Hypnologie*, i., 4, p. 97, 1890).

CEREBRUM: CINGULUM.

59. Course of the Fibres of the Cingulum and the Posterior Parts of the Corpus Callosum and of the Fornix in the Marmoset Monkey. BEEVOR (*Proc. Roy. Soc.*, xlviii, 294, p. 271, 1890).

CEREBRUM: COMMISSURES.

60. Substitution in the Brain. GALLERANI (*Il Morgagni*, xxxii., 9, p. 124, 1890).
61. Physiology of Commissural Fibres. STEFANI (*Arch. per le Sci. Med.*, xiv., 10, p. 243; *also Arch. Ital. de Biol.*, xiii., 2, p. 350, 1890).

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62. Absence of the Grey Commissure of the Third Ventricle and Concomitant Psychic Peculiarities. DE MACEDO (*Bull. de la Soc. d'Anthrop.*, xii., 4, pp. 513 and 541, 1890).

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63. Bilaterality, Associated Movements, Functional Relations of the Corpus Callosum. MOTT (*Brit. Med. J.*, 1890, p. 1124).
64. Tumour of the Corpus Callosum. BERKLEY (*Amer. J. of the Med. Sci.*, 6, p. 578, 1890).
65. Absence of Corpus Callosum. DUNN (*Guy's Hosp. Rep.*, xlvi., p. 117, 1889).

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66. Fornix and Structures connected with it in the Brain of Man and other Mammals. HONEGGER (*Receuil de Zool. Suisse*, v., 2, p. 201, 1890).

INTERNAL CAPSULE.

67. Arrangement of the Excitable Fibres in the Internal Capsule of the Bonnet Monkey. BEEVOR AND HORSLEY (*Proc. Roy. Soc.*, xlvii., 286, p. 21).

OPTIC THALAMUS.

68. The Nuclei of the Thalamus in the Rabbit. NISSL (*Tagebl. d. 62, Vers. deutscher Naturf. in Heidelberg*, p. 509, 1890).

MID-BRAIN.

69. The Connection between the Posterior Corpora Quadrigemina and the Auditory Nerve (further contribution). FLECHSIG (*Neur. Centralbl.*, ix., 4, p. 98, 1890.)

70. The Fibre Systems of the Mid-Brain. EDINGER (*Neur. Centralbl.*, ix., 13, p. 411, 1890).

CORPORA QUADRIGEMINA.

71. The C.Q. as Centre of reflex Ocular Movements. BASSERI (*Ann. di Ottalm.*, xviii., 6, p. 520, 1890).

SUBSTANTIA NIGRA.

72. Minute Anatomy of the Substantia Nigra Soemmeringii. MINGAZZINI (*Atti della R. Accad. dei Lincei*, v., p. 36, 1890).

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73. Atrophy of Fibres in the Cerebellum. MEYER (*Zeitschr. f. Psychiatrie*, xlvi., 5, p. 664, 1891).

74. The Minute Structure of the Cerebellum. KÖLLIKER (*Zeitsch. f. Wissench. Zool.*, xlix., 4, p. 668, 1890).

75. The Physiology of the Cerebellum. LABORDE (*C. R. Soc. de Biol.*, 1890, p. 46, Jan. 25).

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76. The Minute Anatomy of the Nucleus Arciformis and its relations to the Fibræ Arciformes Externæ Anteriores. MINGAZZINI (*Atti della R. Accad. Med. di Roma*, xv.).

77. Striæ acusticæ and Inferiôr Fillet. MONAKOW (*Archiv. f. Psychiatrie*, xxii., 1, p. 1, 1890).

78. Relations of the Middle Cerebellar Peduncles to the Corpus Restiforme. MINGAZZINI (*Atti d. R. Accad. delle Sci. di Torino*, xxv., 11, p. 542, 1890; *also Arch. per le Scienze mediche*, xiv., 11, p. 245, 1890).

MEDULLA OBLONGATA.

79. The Anatomy of the upward-continuation of the anterior and lateral columns (Vorderseitenstrangreste). AUERBACH (*Virchow's Archiv*, 1 and 2, p. 199, 1890).

SPINAL CORD.

80. Posterior root-fibres which originate in the anterior horn. LENHOSSÉK (*Anat. Anz.* V., 13 and 14, p. 360, 1890).

81. Posterior Roots and Posterior Columns. LENHOSSÉK (*Mitth. a. d. Anat. Inst. zu Basel*, 1890, p. 86).

82. The Degenerations which follow section of the Posterior Roots. ODDI AND ROSSI (*Arch. Ital. de Biol.*, xiii., 3, p. 382).

83. Structure of the Spinal Cord in Mammals. RAMÓNY CAJAL (*Trab. del Lab. Anat. de Barcelona*, p. 1, April 1, 1890).
84. The Minute Anatomy of the Spinal Cord. KÖLLIKER (*Sitz. d. Würzb. Phys.-med. Gesellsch.*, March 8, 1890).
85. Certain Anatomical peculiarities of the Sacral enlargement of the Spinal Cord in Birds. LACHI (*Pisa*, 1889, *Nistri and Co.*).
86. Pyramidal Tracts in the Spinal Cord. LENHOSSÉK (*Math. u. Naturw. Bericht aus Ungarn*, vii., p. 47, 1890).
87. The Anatomy of the System in the Spinal Cord which degenerates upwards. AUERBACH (*Anat. Anz.* v., 7, p. 214, 1890).

SPINAL CORD: PHYSIOLOGY.

88. Case of Localization of a limited lesion of the Spinal Cord from Physiological data. MACEWEN (*Brit. Med. J.*, 1527, p. 775, 1890).
89. Hyperæsthesia after Destruction of Cervical Cord MARTINOTTI (*Arch. f. Anat. u. Phys. Phys. Abth.*, *Supplement*, p. 182, 1890).

CENTRAL GRAY TUBE.

90. The Central Gray Matter. SCHÜTZ (*Tagebl. d.* 62, *Vers. Deutsch. Naturf. in Heidelberg*, p. 517, 1890).

CONDUCTING PATHS.

91. Sensory Paths in the Central Nervous System. EDINGER (*Dtsh. Med. Wochenschr.*, 20, p. 421, 1890).
92. On the Degeneration resulting from removal of the Cerebral Cortex and Corpora Striata in the Dog. LANGLEY AND GRÜNBAUM (*J. of Phys.*, xi., p. 606, pls. xiii., xiv).
93. Double Crossing of Cerebro-spinal Conducting-Paths. UNVERRICHT (*Neur. Centralbl.*, ix., 16 and 17, 1891.
94. Paths of Secondary Degeneration in a case of Injury of the Cervical Spine. MEIGS (*Amer. J. Med. Sci.*, Aug. 1890, p. 128).

NERVES: CRANIAL.

95. Structure and Homologies of the Cranial Nerves of Amphibia. STRONG (*Zool. Anz.*, xiii., p. 598, 1890).

95A. The Cranial Nerves of Torpedo. EWART (*Proc. Roy. Soc.*. xlvii., p. 290, 1890).

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96. Nervous Electro-motor Force used for the Study of the Optic Chiasm. FAVARELLI AND FASOLA (*Ann. d' ottalmologia*, 1889, p. 20).

97. Chiasma Nervorum Opticorum of the Chimpanzee. Möller (*Archiv. f. Anthropol.*, xix., 3, p. 205, 1890).

NERVES : CRANIAL : VI.

98. Real Origin of the Abducent Nerve, and its Relation to the Facial Nerve in Man. Mingazzini (*Gaz. med. di Roma*, xvi., p. 49, 1890.)

NERVES : CRANIAL : VIII.

99. Electric Excitability of the Auditory Nerve. Lumbroso and Coen (*Il Segno*, March, 1890).

NERVES : CRANIAL : X.

100. Action of Constant Currents and of Catheterization (of the Œsophagus) upon the Human Vagus Nerve. Fort (*C. R. Soc. de Biol.*, May 10, 1890, p. 262).

NERVES : CRANIAL : XII.

101. Origin of the Hypoglossal Nerve. Mingazzini (*Aus dem Psychiatr. Inst. der Münchener Univ. Torino-Spandra e Lazzari*, 1891).

REFLEX ACTION.

102. Condition of the Reflexes in Cases of injury to the Spinal Cord, with special reference to the Indications for Operative Interference. Bowlby (*Brit. Med. J.*, p. 1132, 1890; also *Lancet*, p. 1071, 1890).

REFLEX ACTION : TENDON.

103. Tendon Reflex. Sternberg (*Verh. d. IX. Congress f. innere Med.*, 1890, p. 428.
104. Localization of the Patellar Reflex in the Spinal Cord. Sanna-Salaris (*Ann. di Frenatria, Torino*, ii., p. 118, 1890).
105. The Nature of the Tendon Reflexes. Sternberg (*Wiener Klin. Wochenschr.*, 23, p. 248, 1890; also *Dtsh. Med. Wochenschr.*, 25, p. 554, 1890).

REFLEX ACTION : VIRILE.

106. Bulbo-Cavernous Reflex. Onanoff (*C. R. de la Soc. de Biol.*, May 3rd, 1890, p. 215).
107. The Virile Reflex. Hughes (*Alienist and Neurologist*, Jan., 1891).

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108. Effects resembling Paralysis produced by Section of Sensory Nerves. Pineles (*Centralbl. f. Physiol.*, iv., 24, Feb. 28th, 1891).

Meninges.

109. The Form and Size of the intervaginal space of the Optic Nerve in its Relation to the Canalis Opticus. Pfister (*Archiv f. Ophthalmol.*, xxxvi., 1, p. 83, 1890).

110. Certain Characteristics of the Dura Mater. Trolard (*J. de l'Anat. et Physiol.*, xxvi., 4, p. 407, 1890).

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111. The so-called Chalk-vesicles in the Spinal Ganglia of the frog and their connection with the Ductus Endolymphaticus. Coggi (*Anat. Anz.*, v., 6, p. 177, 1890).

Sympathetic System.

112. The Anatomy of the Great Sympathetic in Elasmobranchs and Osseous Fishes. Chevrel (*Thesis. Portiers*, 1890).

113. The Function of the Coeliac Plexus. Lustig (*Beitr. f. Path. Anat., Ziegler*, vii., p. 433, 1890).

114. Extirpation of the Plexus Coeliacus. Peiper (*Verh. d.* IX. *Congr. f. inn. Med.*, p. 497, 1890).

Ganglion, Ciliary.

115. Development of the Ciliary or Motor Oculi Ganglion. Ewart (*Pro. Roy. Soc.*, xlvii., p. 287, 1890).

116. Morphology and Structure of the Ciliary Ganglion, Antonelli (*Giorn. della Assoz. dei Natur. dei Napoli.*, 1890).

Electric Organs.

117. The Numerical Relation between the Nerve Cells in the Lobus Electricus of the Torpedo and their End-organs. Fritsch (*Du Bois-Reymond's Archiv.*, 1 and 2, p. 183, 1890).

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118. Suggestions upon the Significance of the Cells of the Cerebral Cortex. Herrick (*The Microscope*, p. 33, *Trenton*, 1890).

Histology : Grey Matter.

119. The Minute Structure of the Grey Nerve Tissue. Heitzmann (*J. of Nervous and Mental Disease*, xvii., p. 357, 1890).

120. The Division of Ganglion Cells in the Lowest Sections of the Human Vertebral Canal. Hoche (*Neur. Centralbl.*, Feb. 15, 1891, pp. 100-102).

Histology : Fibres.

121. Myelin, Medullated and Non-Medullated Nerves. Gad and Heymans (*Archiv. f. Anat. u. Physiol.*, p. 530, 1890, *Phys. Abth.*).

HISTOLOGY: EPENDYMA.

122. The Nature of the Ependymal Epithelium. MAGINI (*Bull. della R. Accad. Med. di Roma*, xvi., 23, p. 116, 1890).

HISTOLOGY: NEUROGLEIA.

123. The Neurogleia-Framework of the Human Central Nervous System. WEIGERT (*Anat. Anz.*, v., 19, p. 543, 1890).

HISTOGENY.

124. The Neuroblasts of the Oculomotor and Trochlear Nerves. MARTIN (*Anat. Anz.*, v., 18, p. 530, 1890).
125. At what Period do the Expansions of the Nerve Cells of the Spinal Cord of the Chick Appear? CAJAL (*Anat. Anz.*, v., 21, p. 609, 1890).
126. The Development of the Peripheral Nervous System in Mammals. CHIARUGI (*Atti della Soc. Tosc. di Sci. Nat. Proc.*, vii., 1890).
127. Histogeny of the Neurogleia of the Spinal Cord of the Chick. LACHI (*Atti d. Soc. Tosc. d. Sci. Nat.*, xi., 1890, *Pisa*).

DEGENERATION.

128. The Dependence of Degeneration in Peripheral Nerves upon destruction of their Nuclei of Origin. KRONTHAL (*Neur. Centralbl.*, March 1, 1891, pp. 133 to 141).

REGENERATION.

129. Reparative Processes in the Cerebrum and Cerebellum. SAVARELLI (*Riforma Med. Napol.*, 1890, p. 626).
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B R A I N.

PARTS II. & III., 1891.

Original Articles.

ON THE SENSORIMOTOR NERVE-CIRCUIT OF MUSCLES.

BY PROFESSOR A. CHAUVEAU.

This title was suggested to me by a reminiscence of the International Medical Congress, held in London in 1881. During the opening address of the Physiological section by Professor Foster, I was struck by what the speaker said of the manner in which Charles Bell understood the relations between the sensory and the motor nerves, that is, of his conception of the *nerve-circuit*. It is true that this expression could not establish itself, because its adoption would have assumed the existence of an unlikely physiological fact. It is indeed certain that the existent knowledge concerning the nervous system did not allow of the reality of a *nervous circulation*, or of a *nerve-circuit*, in the strict and definite sense of these words.

It is, nevertheless, quite certain that sensory nerves influence the action of motor nerves and *vice versâ*. I confess that I have often been tempted to express the general mechanism of their physiological relations by Charles Bell's locution, which is striking and picturesque. The mention made of it by Prof. Foster made me take the resolution to use it whenever I should give an account of the numerous experiments I have made, at different times, on the functions of the sensory nerves of muscles. This is what I attempt to do in the present paper, which, at the instigation of my friend, Dr. Waller, I have the pleasure to publish in the country and language of Charles Bell—a fact which accentuates the intention to pay thereby a deserved homage to the memory of one of the founders of neurophysiology.

I shall deal in the present essay on muscular nerves only. All muscles are supplied with motor and sensory nerves: though this fact is rarely demonstrable anatomically, it is always so physiologically in health or disease. For instance, it is easily demonstrated in the case of voluntary muscles by squeezing of nerve twigs which spend themselves completely in these organs; such an excitation produces always pain, and sometimes reflex movements, if the subjects have been prepared for the manifestation of such a reaction.

This muscular sensibility has been much studied, even from the psycho-physiological point of view; for it is the primordial condition of what is called the muscular sense. But this property used to be considered as only a case of the general sensory mechanism. Nobody thought of otherwise connecting the sensory with the motor function. One used to say that the contraction excited by the latter utilised, for its regulation, the sensibility given to the muscle by the former. The two nerves were then considered as two isolated physiological systems, acting simultaneously side by side.

Now when, after Charles Bell, we use the expression *nerve-circuit* to designate this arrangement, we imply in some way the existence of one and the same conducting system, composed of two parts, centrifugal and centripetal, intimately connected together. Can the study of the sensory and motor nerves of muscle be influenced in its results, if these conductors are thus united into a single physiological system? One would be tempted to expect from this union nothing but a difference in the mode of presenting facts. But I hope to show that both the acquisition and the interpretation of facts, gain by the adoption of Charles Bell's view of the nerve-circuit.

And first: What anatomical idea can be framed of this circuit? We know absolutely nothing of the relations existing between the terminal filaments of the sensory nerve tubes, and the muscular tissue itself. Still there is room for believing that these tubes have no direct relation with muscular elements, and for supposing that they contribute in forming the pre-terminal anastomoses or networks of the motor nerves in the muscular system. In the case of voluntary muscles, be it so or otherwise, it is easy to conceive as a circuit the sensorimotor nerve apparatus of the muscular fasciculus.

Under the hypothesis of a juxtaterminal unity of the two kinds of nerve fibres, we may picture to ourselves the circuit in the following manner among others: starting from the motor cell, which is the origin of the centrifugal tube, it would reach through the latter the anastomosis next to the terminal plate. Thence through the centripetal tube supposed to end in this anastomosis, it would return to the sensory group of cells in the medullary centre, finally merging into its starting point through the commissure connecting the motor and sensory cellular groups.

In the unlikely case that special sensory endings should be discovered along the muscular fasciculus, the circuit would be completed through the muscular substance itself, between the motor and sensory nerve endings. I consider this case as unlikely for two chief reasons: the first is, that in voluntary muscle (with which we are at present concerned), the true nerve-endings are now well-known, and universally considered to be motor only (terminal plates and ramifications, Doyère's eminences). The second, which would be sufficient in itself, is that in very short muscles, each primitive bundle carries but one such nerve-ending, very rarely accompanied by a small satellite attached to the same cylinder as the chief organ. But I wish to guard myself against any definite conclusion upon this difficult point, which calls for further histological investigations.

The concept of a nerve-circuit is still more admissible in the case of visceral muscles. Every kind of nerve fibre supplying them ends in networks studded with nerve cells, which, perhaps, send fibrils in to the unstriated muscular elements.

If the nerve-circuit is really constituted as just stated, the elementary muscular organs are not placed in its course, but are merely *tangent* to it, either directly or through terminal structures, such as plates, fibrils, &c. This remark is important, because this tangential disposition complicates the problem of neuro-muscular interconnections. But since our present knowledge does not allow us to explain even the transmission of excitations from nerve to muscle, its mechanism does not become less intelligible by the admission that the nerve-endings do not directly and separately connect the muscles to the two kinds of nerves, but to the common circuit formed by the union of these nerves near their peripheral end.

How shall we proceed to study experimentally, by laboratory methods, the properties and functions of the two portions of the sensorimotor circuit? We have to determine the influence of each upon muscular action. Hence we shall first study the results of excitation of each portion before section; then after division those of excitation, both of the central and of the peripheral end. We shall next investigate the effects of interruption in the centrifugal, and in the centripetal portion; and finally, compare the effects obtained in each case.

Such is the programme of all analogous experiments. Let us add to it, if possible, a study of the commissures which, in the cerebro-spinal axis completes the circuit by connecting together the central ends of the centrifugal and centripetal paths. But how shall we fulfil this task? And where shall we find suitable organs to work upon? We require organs in which the sensory be quite distinct and isolated from motor fibres. I have, hitherto, found but two such, one belonging to the locomotor system, the other to the visceral; but these have sufficed to carry out my scheme with reference to the nerve-circuit of both sets of muscles.

1.—ON THE SENSORIMOTOR NERVE-CIRCUIT OF VOLUNTARY STRIATED MUSCLES.

The nerve supplying the sternomastoid muscle of the horse enters it through a slit close to the terminal tendon. Following the nerve upwards from this point it is seen to divide. The larger branch joins the external branch of the spinal accessory, of which it forms one of the divisions. The other is supplied by the inferior branch of the second cervical pair. The disposition of these two nerves is generally simple; sometimes, however, they become plexiform at their point of union.

The first is the motor branch of the muscle; the second is purely sensory. Squeezing of the combined nerve near its point of entrance produces pain, but after division of the second branch gives rise to no sign of sensation whatever.[1] I may add that excitation of the peripheral end of this branch never gives rise to contraction of the muscle.

Thus we have here present the fundamental anatomical condition required by the experimental and comparative study of the influence excited on a given muscular action by the two portions, centrifugal and centripetal, of its nerve circuit. The operative procedure required to expose them is so easy and simple that it need not be described here.

I shall not enter upon any details upon the actual experiments made. It will be enough to describe the results, which have been uniform, and of the exactness of which I am perfectiy satisfied.

A.—*Comparative excitation of the centrifugal and centripetal portions of the sternomastoid nerve-circuit.*—This excitation has been carried out sometimes by squeezing, sometimes by single or repeated induction shocks.

(*a.*) *Excitation of the two branches, motor and sensory, without interruption of their continuity.*—Single induction shocks, of minimal strength, are suitable for this experiment. The two nerve branches must remain in contact with the subjacent structures (*M. Ant. rect. capitis major*) in order to avoid any electrical derivation. They are thus excited at the electrodes only, the points of contact being the only ones where the current has enough density, or where the potential is high enough, to act upon the nerve fibres.

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[1] On a donkey I observed, after division of the second cervical branch, the persistence of some sensibility in the combined nerve. *Post-mortem* dissection however, showed the existence of a filament from the first cervical pair. I cannot say whether this disposition is usual in the donkey, nor whether it may not be exceptionally present in the horse.

(1.) *Centrifugal part of the circuit (motor branch.)*—I have never observed along with the muscular contraction obtained any clear sign of pain, even on marked increase of the current strength. I had previously noted the difficulty of obtaining, in the horse, signs of recurrent sensibility on exciting the spinal accessory too near its point of origin.

(2.) *Centripetal part of circuit (sensory branch.)*—The minimal excitation thus necessary to obtain muscular contraction does not generally provoke any reaction when applied to the centripetal branch; the current strength must be somewhat increased. Strange to say the first effect observed, in certain subjects, has been a contraction of the sternomastoid. It was then difficult to discriminate between the effects of the centrifugal and that of the centripetal branch; the latter, however, was not quite so immediate as the former. Stronger excitations bring about additional phenomena, of which we shall speak further on.

I need not say that the contraction obtained on excitation of the sensory portion is of reflex origin, and constitutes a remarkable phenomenon by its exclusive localisation of the muscle to which the nerve-circuit belongs. The following facts bear still further upon this important datum.

(*b.*) *Excitation of the motor and sensory branches, interrupted in their continuity.*—Let us now investigate what takes place when the peripheral and central ends of each of the two branches has been divided.

(1.) *Peripheral ends.*—Excitation of the motor branch still calls forth contraction; that of the sensory branch has no effect whatever.

(2.) *Central ends.*—The results are opposite; excitation of the motor branch having no effect, whilst that of the sensory branch is followed with contraction of the muscle. Sometimes, however, the reflex contraction, even when the stimulus is very slight, is accompanied with more generalised movements which are a sign of pain. Still the latter reaction does not usually appear quite so quickly as the localised reflex contraction, and thus the two can be discriminated. But the reflex contraction following an excitation of the sensory nerve is not always localised in the sternomastoid. On increasing the intensity, it may extend to all the muscular apparatus innervated by the external branch of the spinal accessory, and invade other regions on the same side of the body. In the case of strong stimuli it may even generalise itself on the opposite side of the body. I have been able to clearly observe this effect in many apathetic, unimpressionable subjects, easily bearing pain without reacting by any general voluntary and disorderly movements.

(*c.*) *General review of the conclusions reached by excitation of the nerve-circuit in muscles of animal life.*—These results favour the nerve-circuit theory of the relations between the sensory and the motor nerves of muscles, and may be summed up as follows :—

(1.) Stimuli applied to the nerve-circuit travel in a centrifugal or a centripetal direction according to the part excited.

(2.) Whatever part be influenced, contraction of the muscle follows, directly in the case of the centrifugal, indirectly through the nerve centres in that of the centripetal arc of the circuit.

(3.) Interruption of the circuit beyond the excited point, that is in the path through which the stimulus has to travel, prevents the latter from reaching the muscle and make it contract.

(4.) Whatever be the strength of the excitation applied to the centrifugal arc remains localised in the muscle which it has reached directly, and never produces anything beyond its contraction.

(5.) When the stimulus applied to the centripetal arc is very weak, it is wholly reflected in the central cellular groups upon the centrifugal portion of the circuit, and then produces a contraction of the corresponding muscle only.

(6.) When the excitation of the centripetal branch is stronger it may diffuse itself in the medullary centre, and provoke a contraction of more numerous muscles, among which the first affected are always those which are innervated by the same group of motor cells as the muscle whose nerve-circuit is being excited.

These first results of excitation of nerves supplying voluntary muscles encouraged the study of the effects of *suppression* of the conductibility of the nerve-circuit. Experiments made in that direction, however, did not yield the expected results; still they are interesting, as we shall see when we discuss their physiological significance.

B.—*Influence of the interruptions in the nerve-circuit of the sternomastoid.*—Transverse section, with or without excision, of the motor and sensory branches of the nerve supplying the sternomastoid, is as simple an operation as their excitation. I had intended to study its influence upon both muscular nutrition and normal contraction. I have, however, not been able to fully investigate the latter point.

(*a.*) *Effects produced upon the physiological voluntary contraction of the sternomastoid by interruption of its nerve-circuit.*—The sternomastoid of the horse acts chiefly as a flexor of the head. In order to bring it into play a wisp of hay is placed in the manger fixed rather high. The animal raises its head, seizes the hay with its incisors, and in order to bring it down suddenly flexes the head upon the neck. The exploration with the eye, or rather with the hand, of this region shows what takes place, at least whether the muscle contracts or not.

It was hardly necessary for me to investigate again the well-known effect of section of the motor branch of the nerve; the whole interest of the experiments centred upon that of section of the sensory branch. For never, to my knowledge, had the results of total and exclusive sensory enervation of a muscle been observed before. In previous experiments, as we shall have occasion to see presently, the abolition of sensibility affected a whole region, such as a whole posterior limb, and included all tissues, skin as well as muscle. Here we abolished nothing but the function of the sensory nerve supplying the contractile organ, and the conditions in which we were placed could not be more favourable for the comparison of the two kinds of nervous functions. The results obtained have been identical, both in cases where the experiment was made extemporaneously and where the subject was observed for some weeks afterwards. Whilst the muscle which has undergone motor enervation remains absolutely paralysed, that which has been subjected to sensory enervation continues apparently to contract normally. For instance, in a subject whose motor nerve has been cut on the left, the sensory on the right, the left sternomastoid is felt to remain flaccid, whilst the right becomes rigid on contraction, when the animal flexes its head on pulling hay out of the manger.

I have never met with any exception to this rule, hence we may assert that, contrary to what happens on section of the motor branch, section of the sensory branch does not abolish movement. But we may not go beyond this somewhat vague conclusion, for we do not know whether movement, though not abolished, is not in some way altered cr modified. Notwithstanding repeated and careful observation of subjects in whom unilateral sensory enervation had been performed, I failed to obtain evidence of any alteration of function. Flexors of the head are numerous, and easily supply each other's place; moreover, direct observation of the muscular action by palpation is not a delicate enough method for the analysis of that function.

Hence we reach this indefinite conclusion, that after section of its sensory nerve the sternomastoid muscle does not lose its voluntary contractibility, but that it is impossible to say whether its voluntary movement is not more or less disturbed.

This result appears to be in complete contradiction with the facts observed by Claude Bernard after section of a series of lumbar sensory roots in the dog. The corresponding hind limb has its movements disturbed, and may even become incapable of supporting the body. Now this disturbance is not due to cutaneous anæsthesia, for it never follows section of the skin nerves.[1] The paralytic phenomena observed in the dog are obviously due to the destruction of sensibility in the leg muscles through interruption of the centripetal branch of their nerve-circuit.

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[1] Section of the cutaneous nerves of the extremities has often been performed in order to determine their functions in locomotion and station. I have performed the experiment on some horses and many pigeons. In every case I found that complete sensory enervation of an extremity does not appreciably disturb these functions. My experiments on pigeons deserve mentioning; they were carried out on tame specimens, very easy to observe. I used to make one of the feet completely anæsthetic by cutting around the tarsal articulation the four nerves supplying the toes, which could thus be

The accuracy of this explanation may be doubted because the conditions of the experiment are not simple. The traumatism is serious, and it is not always easy to appreciate the real motor disturbance in a dog's hind leg from the attitude given to the limb. I, however, fully adhere to it, and hope further on to demonstrate that there is no contradiction between the facts to which it is applied, and those furnished by my own experiments. The discussion of the part played by the sensory nerves in the muscular functions of the leg, will show why the necessity of their intervention was not manifested in my experiments on the sternomastoid of the horse.

(*b*.) *Effects produced upon the muscular nutrition by interruption of the nerve-circuit.*—Among the animals upon which these experiments have been performed some were preserved for several weeks, others even till complete cicatrisation of the extensive wound. After sensory enervation the muscle always remained contractile, though it occasionally appeared as if its contractions became somewhat less vigorous as time elapsed. Was this a fact, or was I the victim of an illusion, natural enough under the circumstances? I resorted to post-mortem examination of the tissues to find out whether sensory enervation did not involve corresponding muscular degeneration, and examined two cases, six and seven weeks respectively, after the

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pricked, squeezed, cut, burnt, without giving rise to the least sign of sensation. When the animal is placed upon the ground after the operation, it does seem to hesitate in the use of the enervated foot. This hesitation consists, at the moment of starting on to walk, in a repetition of the movement of placing the toes upon the ground. The animal looks as if astonished not to feel this contact, but once started it uses both feet equally freely.

It is especially during sleep that these animals show how completely the mechanism of station is independent of cutaneous sensibility, for they roost indifferently upon the normal and upon the enervated foot. I used to place my subjects during the night on a perch at my bed side, and every time I awoke I observed them by means of an arrangement for the sudden projection of light, before they had had time to change their position on being aroused. I had facilitated the observation by previously colouring differently the two feet. Persevered in during several months these experiments always gave uniform results; the animals, during sleep, used to stand about as often on the anæsthetic as on the normal extremity.

operation. Now, in neither did I observe any such degeneration. The muscle did not appreciably differ from its normal fellow in either colour, weight, and histological characters. I admit that these experiments do not prove that sensory enervation does not have any influence upon muscular nutrition, for in very old horses, such as were used by me, degeneration after total enervation always takes place very slowly. However, I must add that forty-seven days after motor enervation only, unmistakable changes were apparent in the muscle, which was paler than its fellow, and whose fasciculi were infiltrated with fatty granulations.

If, therefore, it is not possible to affirm that the sensory branch of the sternomastoid nerve exercises no action upon muscular nutrition, it is at least possible to affirm that it has infinitely less influence upon it than the motor branch. Further researches, made under more favourable conditions, must be carried out in order to settle this point.

C.—*Summary.*—Interruption of the sensorimotor nerve-circuit of muscles of animal life, has twofold effects upon muscular nutrition and function.

Upon nutrition; (1) section of the motor branch always causes atrophy and fatty degeneration; (2) section of the sensory branch does not appear to disturb nutrition, but this assertion requires to be controlled by further researches.

Upon function; (1) section of the motor branch immediately paralyses the muscle; (2) that of the sensory branch does not suspend response to voluntary stimuli; (3) voluntary movement of the muscle after its sensory enervation appears to be normal, though it is impossible, owing to the nature of the experiments, to exclude all possibility of slight disturbances; (4) these results do not appear to conform to those obtained by Claude Bernard, in the total sensory enervation of the dog's hind leg, but the contradiction is apparent only. The conditions under which he experimented are not so simple as those obtaining in our researches. We shall return to this point, which we mention here only to guard against any premature conclusion as to the non-participation of the centripetal branch of the circuit in the muscular acts of locomotion. The results described in the next paragraphs justify our reserve on this point.

II.—ON THE SENSORIMOTOR NERVE-CIRCUIT OF INVOLUNTARY STRIATED MUSCLES.

In order to determine the effects of a rupture in the centripetal arc of the circuit on the physiological movement of the muscular organ (1) we should be able at will and with certainty, to excite that movement; (2) which must be always identical and known in all detail; and (3) be of easy observation.

Now all these conditions are united in an automatic, always similar, and well-regulated act, that of œsophageal deglutition. And very fortunately the observable (cervical) portion of this tube, receives in the horse its motor and its sensory supply from two different sources. I shall recapitulate the description I have given elsewhere of this arrangement.

The nerves of the cervical part of the œsophagus do not present any uniform arrangement in all mammals, but are arranged according to three types. In the rabbit as in man, motor and sensory fibres are intimately mixed in the recurrent nerve, and follow an ascending direction. In the dog, they are equally closely connected together, but are supplied by the pharyngeal nerve, and follow a descending course. In the solipedes they are completely separated, and take a descending and an ascending course respectively.

The descending nerves lying superficially on the sides of the œsophagus or embedded in its substance, can be traced from the pharynx into the chest; and arise from the pharyngeal and external laryngeal nerves. The evidence showing that these are the motor nerves will be stated further on.

The ascending nerves are fine twigs supplied by the thoracic portion of the vagus or the recurrent nerve. They are themselves recurrent œsophageal nerves, disposed in no constant manner. Some are very long, sometimes reaching up to the origin of the tube; others, shorter, supply its thoracic extremity, and are of chief interest to us. They are all centripetal nerves, as I have shown elsewhere, and shall point out again presently.

The ramifications of these sensory nerves obviously do not all go to the mucous membrane; some supply the muscular coat, forming, with the motor branches, the nerve-circuit of the œsophageal muscle in the cervical region.

Since, in the great majority of our present experiments, we cannot avoid acting on the sensory branches both of the mucous œsophageal membrane and of the nerve-circuit, we must first determine the part played to the sensibility of that membrane in the movements of deglutition, in order to eliminate from our results those not due to excitation or rupture of the muscular nerve-circuit.

Now we know that the functions of the mucous membrane of the cervical œsophagus do not influence the physiological movement of this portion of the tube. I showed that, in the horse, as in the rabbit and the dog, regular coördinated peristalsis occurs from the pharynx to the thorax during deglutition *à vide*. In the rabbit it is easy to demonstrate the fact, simply by dividing, with scissors, the œsophagus at its origin. The nerves being all ascending need not be considered. When, now, a movement of deglutition is set up, by seizing with a forceps the œsophageal stump that remains adherent to the pharynx, and pulling the latter backwards, one sees in the isolated œsophagus a continuation of the peristaltic movement, as if the organ had a bolus to convey. Sensory excitation of the mucous membrane by the bolus does not, therefore, take a direct part in the provocation and execution of this movement, at least in the cervical pertion of the œsophagus.

In order to demonstrate this fact in the horse more care is needed, since we must avoid injuring the motor nerves adherent to the œsophagus. A wide slit is made at the origin of the latter, and a ligature involving the mucous membrane only is applied below the aperture. When now the animal is eating, the bolus can no longer penetrate into the œsophagus, but escapes at the outlet of the pharynx. Regular peristaltic movements are now observed throughout the cervical length of the tube; only the strength of contraction goes on diminishing from top to bottom, whence we conclude that the presence of food in the tube excites it only to give to its contractions the power necessary to drive forwards the alimentary bolus.

This proof of the non-intervention of mucous sensibility in the provocation and execution of coördinated deglutitional movements of the œsophagus, is quite peremptory. I will add still another, based upon an experiment showing that the excitation of the nerves of the mucous membrane does not cause any reflex movement of the œsophageal muscle. The value of this experiment will become evident when it shall be compared with a corresponding one, made upon the ascending branches which supply sensory filaments to both membrane and œsophageal nerve-circuit.

The experiment is as follows: the œsophagus having been rather extensively exposed on the left side, near the thorax, the muscular tube is slit longitudinally in the upper plane, care being taken not to injure the nerve branches. The mucous tube is thus brought into view, the nerve filaments supplying it being conspicuous on its surface. Excitation of these purely mucous filaments may easily be carried out by means of fine electrodes connected with an induction coil. Now I have never succeeded in thus producing the least reflex contraction[1] of the œsophageal muscle.

This preliminary discussion has prepared us for our experimental study. We know that by acting on the descending and ascending nerves, we act upon the centrifugal and centripetal branches of the circuit respectively. We may now proceed to observe, with perfect safety, the effects of excitation and interruption of the œsophageal nerve-circuit.

(*a.*) *Influence on the physiological coördinated œsophageal contraction, of interruption of the centrifugal and*

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[1] This does not mean that the presence of an alimentary bolus arrested in the œsophagus, or of a foreign body introduced through a slit into it, cannot call forth more or less insensible contractions, alternately suspended and resumed, which aided by thoracic aspirations, end in causing a slow progress towards the stomach. But this is a phenomenon wholly different from true deglutition.

centripetal arcs of the nerve-circuit.—Let us take first the motor or centrifugal portion. It is easy enough to divide all the descending nerves at the origin of the œsophagus, and to observe the latter through long incisions made along the jugular furrow. One sees then that the cervical portion of the tube no longer contracts on deglutition. The bolus projected into the œsophagus by pharyngeal contractions soon come to a standstill, and remains stationary until another bolus pushes it down. This result was expected; still we must mention that in order to obtain it, we must divide all the descending nerves on both sides. Strange to say, unilateral section does not sensibly modify œsophageal deglutition: as if the whole of the œsophageal muscular elements were controlled by the nerves on each side independently, and excitation from the right propagated itself to the left, and *vice versâ*. Unilateral section of the motor nerves may deprive some muscular fasciculi of every supply, and so paralyse them; but the rest remain fully innervated. Thus the process of deglutition may be weakened—a fact difficult to ascertain—but is impaired neither as to its regularity nor its efficacy.

As to the effects of interruption of the centripetal arc of the œsophageal nerve-circuit, it is not easy to divide all the ascending œsophageal branches; I was so led to divide the trunk itself from which they arise (viz., the vagus) below the point of origin of the motor branches.

Now after section of both vagi, though they contain no motor fibres to it, the cervical portion of the œsophagus displays profound disturbances in the coördinated movements of deglutition. These troubles are various. Sometimes the muscular tunic is paralysed, as after section of the motor nerves, but never in its upper third. The loss of power begins in the middle third, and becomes quite apparent in the inferior portion, where arrest of alimentary boli takes place either temporarily, or permanently, thus leading to obstruction along the whole œsophageal tract. Sometimes—chiefly in donkeys—the œsophagus becomes as it were ataxic, and contracts irregularly and almost simultaneously along its whole length. This happens especially when the animal coughs, or when it is obliged to make some effort at deglutition.

Unilateral enervation appears at first sight to be as unefficatious in the case of the sensory, as it is in that of the motor, œsophageal nerves; for after section of either of the vagi the peristaltic contraction of the tube seems very little affected, when it is not quite regular. But the sequel shows that this operation profoundly modifies the process of deglutition. Arloing and Tripier found that, seven times out of twelve, in the donkey, division of one vagus is followed by complete obstruction, by food, of the œsophagus, from the pharynx down to the cardiac orifice, with or without filling and distension of the stomach. No doubt that in these cases the divided nerve has greater influence than its fellow upon the motor innervation of the terminal portion of the œsophagus, which becomes plugged, and thus arrests further boli still propelled by the upper portions of the tube. But division of the vagus having partly interfered with the action of the motor nerves upon these portions also, this disturbance eventually becomes apparent by œsophageal incapacity to force into the stomach the alimentary plug retained in the terminal portion of the canal.

Thus interruption of the nerve-circuit of the œsophageal muscle always determines serious disturbance in its physiological function: (1) Complete abolition if it occurs in the motor arc; (2) Sometimes complete abolition, sometimes motor incoördination, if it occurs in the sensory arc. Hence this centripetal portion of the circuit plays an important part in the normal action of the œsophagus.

(*b*.) *Comparative excitations of the centrifugal and centripetal portions of the œsophageal nerve-circuit.*—We excited in every case with rapidly intermittent induction currents, at the points previously selected for division of the nerves. The stimuli were applied either to the intact branches, or to their central and peripheral ends after section. We shall consider the results obtained in the latter case only, which are the most interesting.

(1.) *Excitation of the centrifugal portion (descending nerves.)*—Excitation of the peripheral end brings forth tetanisation of the cervical part of the œsophagus.

Excitation of the central end remains without any effect. Still I am not certain whether it did not provoke in the animal a sort of attention, if not of anxiety, as if a few sensory fibres were contained in these nerves. These fibres possibly supply the upper part of the œsophagus and inferior pharyngeal constrictor muscle; and this would explain why the action of the upper part of the canal is not much disturbed, at least at the outset, by division of the vagus in the middle of the neck.

(2.) *Excitation of the centripetal portion (trunk of vagus in the mid-cervical region.)*—The experiment is quite easy, but observation of the results is made sometimes difficult on account of the numerous other nervous elements excited, besides those of the nerve-circuit.

Such difficulties do not arise when the peripheral end is excited, for the stimuli must always be weak, too weak to produce stoppage, or even slackening, of the heart. We then obtain contractions of the stomach, and of the intrathoracic portion of the œsophagus; the latter becoming manifest by a dragging, front to back, of the cervical portion which never itself displays any contractions.

Difficulties arise on exciting the central end, which process is accompanied with cough, nausea, forced inspirations, &c. Still one may generally observe what takes place in the œsophagus, especially if the stimuli are weak and short. Total tetanisation, more or less permanent and regular, simultaneous (at least to the eye) at all points, is always determined. This tetanisation appears after a certain delay; but is otherwise identical with that obtained by exciting the peripheral end of the centrifugal nerves.

The circular conduction of excitations is thus more clearly demonstrated here than in the corresponding experiment on the sternomastoid nerve-circuit.

(*c.*) *Elimination of the disturbing condition contained in these experiments.*—Though these results are clear enough, I have tried to reach the isolated centripetal fibres of the œsophageal circuit, that is the ascending branches which contain them. I thus hoped to eliminate all concomitant effects due to excitation of other elements of the vagus.

Though the task was laborious and difficult, I succeeded in obtaining perfectly unobjectionable results.

I have already stated that the portion of the cervical œsophagus, which is most influenced by section of the vagus at the stated spot, is that near the thorax. In order to observe the canal in that region and operate on its centripetal nervous twigs, it is necessary to make on the left side (the œsophagus being in the horse, strongly deviated to the left and placed by the side of the trachea) a long incision parallel to the jugular vein, and carried as low down as possible. It is in the lowermost part of the wound that we must look beneath the œsophagus and against the trachea, the ascending branch containing most of these twigs. Electric exploration is used in this search, to reveal the presence of the nerve by the œsophageal contraction it produces.

This contraction is not directly provoked, for if the nerve thus found be divided excitation of the peripheral end has no effect, whilst that of the central end brings about the result just as that of the intact nerve.

In this case the œsophageal tetanisation is the only phenomenon observed ; the animal does not give any sign of pain, nor of cough or nausea, even on strong stimulation. The latter is, in any case, wholly reflected upon the centrifugal portion of the circuit, and thus brought to bear upon the muscle it supplies.

Several times, after finding this ascending branch. I slit up the muscular coat of the œsophagus in order to test the properties of the sensory fibres it supplies to the mucous membrane. The constant negative results of excitation of these fibres contrasted with those obtained by stimulating the branch itself.

Interruption of the continuity of this same branch has effects corresponding to those of its excitation. After its division I observed accumulation of food in the lower cervical tract of the œsophagus, the muscular layer of which was more or less paralysed.

To sum up, the results of the experiments just described confirm those previously described as following excitation or division of the vagus trunks. I will add that the left vagus gives off most sensory branches to the cervical œsophagus, and more particularly the ascending branch on which the last experiments were made.

(*d.*) *Summary of results of experiments in the sensorimotor œsophageal nerve-circuit.*—(1.) The well coördinated movement of œsophageal deglutition lends itself admirably to the study of the respective influence upon muscular motility of the centrifugal and centripetal elements of the nerve-circuit. (2.) In the horse this study is made possible in the middle, and more especially the lower cervical portion of the œsophagus, by a peculiar disposition of the nerves. These portions receive their motor (descending) and their sensitive (ascending) nerves from two points of the vagus. It is thus quite easy to act separately upon these two branches of the nerve-circuit. (3.) Section of the centrifugal fibres produces complete motor paralysis. (4.) Section of the centripetal fibres always markedly disturbs the motor function, causing sometimes transitory and irregular paralysis, sometimes a peristaltic incoördination that interferes with the normal process of deglutition. (5.) Electrisation of the centrifugal arc of the circuit produces œsophageal tetanisation. (6.) Electrisation of the centripetal arc produces tetanisation, but after an appreciable delay. (7.) Tetanisation never occurs when, after division of the nerves, we excite the central end of the centrifugal arc or the peripheral end of the centripetal arc. (8.) We find here, therefore, a justification of Charles Bell's view; and the centrifugal and centripetal nerve fibres, connected together at their extremities by the original cells and their commissures or by the preterminal network, constitute a real circuit. Whenever this circuit be broken there is abolition or marked incoördination of the normal muscular action. Wherever it be excited there is muscular contraction. Finally, wherever it be interrupted during excitation the muscular effect continues or ceases, according as the division occurs above or below the point excited, that is beyond the course of the excitation or within that course itself.

(*e.*) *Concerning the intervention of the mechanism of the nerve-circuit in the function of the œsophageal muscle.*—

I have a remark to make concerning the intimate mechanism of this process. We have to deal with an automatic movement entirely withdrawn from the influence of the superior nerve centres. The action of the centripetal fibres on movement cannot, therefore, be explained by attributing to them the part of intermediaries between the muscle and the psycho-physiological centre for the muscular sense. In the present instance coördination depends only upon the action of the cell groups at the origin of the centripetal conductors. It is difficult to picture to oneself the intimate mechanism through which these sensory cells influence muscular action. But it has been well established that in order to act, these cells must be connected with the muscle through the sensory fibres, and with the motor fibres, through the original cells of these fibres in the nerve centres. A circular propagation of nervous influence is thus implicated, according to Bell's conception of a nerve-circuit.

III.—ON THE CAUSES OF THE DIFFERENCE BETWEEN THE RESULTS OF EXPERIMENTS ON THE STERNOMASTOID AND ON THE ŒSOPHAGEAL NERVE-CIRCUITS.

The two preceding sets of experiments have given us generally analogous results, which, however, seem to differ on an important point. Interruption of the centripetal arc deeply alters the physiological action of the œsophageal muscle, but appears not to do so in that of the sternomastoid. This difference deserves closer study. We stated that we could not affirm that the sternomastoid, after its sensory enervation, fully preserves its normal function. At any rate, it is clear that if its action is disturbed this disturbance cannot be compared with that of the œsophageal muscle. One might at first be tempted to attribute this difference to the fact that the two muscles are not of the same nature. Though both striated, one is an automatic organ, not influenced by the psycho-physiological centres, the other is a part of the voluntary locomotor system, influenced by these centres. Perhaps this system may work regularly in the absence of continuity in the centripetal arc of its nerve-circuit, whilst this continuity is indispensable to the normal action of the striated or smooth visceral muscles, such as the œsophagus.

But this vague explanation is negatived by the above mentioned experiments of Claude Bernard, which demonstrate the necessity of continuity in the centripetal arc, for the normal action of muscles subjected to the higher centres. I do not believe that this conclusion be controvertible, but why then did not my experiments on the sternomastoid have the same result?

In Bernard's experiments it was necessary to divide all the sensory roots of the limb in order to deeply disturb locomotion, that is the coördinated and synergic contractions of the numerous muscles involved in standing and walking. If some of the roots escaped the animal preserved the use of its limb for these two actions. Let us suppose that among the nerve roots those only supplying one muscle, say the extensor of the toes, have been divided. Would its action have been impaired, or would it not rather have shown as little disturbance as the sternomastoid in our experiments? This question may perhaps be answered some day from physiological or clinical data. In the meanwhile the facts of the case seem to admit of another explanation.

It is certain that in locomotion the simplest movement requires the cooperation of numerous muscles, the contractions of which must be regulated, combined, coördinated. These muscles must be connected through their nerve circuit to the medullary groups of cells to which impulse is given from the higher centres. We may well admit that all these solidary organs are involved together in the willed movement, even those whose centripetal, that is unessential, nervous arc is broken.

Such is the explanation I should give of the phenomena observed by us in the sternomastoid. It is associated with other muscles in its motor functions, which are thus connected through their nerve circuits, with the same groups of nerve cells. Each muscle must always be connected with the motor group, but it is not necessary that every one communicate with the sensory group, for those whose nerve circuit is intact are sufficient to bring about the action of all.

This is, no doubt, also the case with visceral muscles. Thus the muscular œsophageal layer is not a simple, but a complex organ, made up of numerous muscles. The nerve circuit of most of them is interrupted by division of the vagus or of the ascending nerves. Some still incomplete experiments lead me to believe that sensory enervation affecting but a very small number of œsophageal muscular bundles would not perceptibly disturb their function. To sum up, I think that the action of the centripetal arc obeys the same laws, both in voluntary and involuntary muscles.

IV.—ON THE CONNECTIONS BETWEEN THE TWO PARTS OF THE SENSORIMOTOR NERVE-CIRCUIT OF MUSCLES IN THE CENTRAL NERVOUS SYSTEM.

The physiological study of the cerebro-spinal connections between the central extremities of the centripetal and centrifugal arcs constitutes perhaps the most interesting and important point in the question before us. Unfortunately they are beyond the reach of experimentation, and can hardly be excited, or interrupted so as to break at their level the continuity of the nerve circuit. These connections lie deeply along the whole medullary axis; they consist of motor and sensory cell groups, which give rise to the corresponding centrifugal and centripetal nerve roots, and of the fibrillar or cellular elements binding the cell groups together, which constitute the true connections between the two systems.

In the absence of a special experimental study of these connections, we may utilise a certain number of collateral data of physiological and pathological origin. But I shall confine myself to very brief remarks on the excitation and suppression of these nerve elements.

(1.) *Excitation of cerebro-spinal cellular and fibrillar elements at the origin of peripheral nerve fibres.*—Concerning the excitability of the cells, which are the real origin of the motor fibres, I observed in a recently killed horse the results of their excitation. I used weak induction currents applied through fine electrodes held near each other, and also scraping with the point of a needle. I shall mention only the clearest facts, elicited by excitation of the nuclei in the fourth ventricle, of the seventh and twelfth pairs. The movements produced in the facial and lingual muscles are identical with those following on the direct excitation of the corresponding nerve roots. When the excitation is spread over the whole cell group all the muscles are tetanised; when it is less strong and localised by maximum approximation of the electrodes, contraction occurs in those muscles only that are innervated by the region excited.

These facts show that both the intramedullary and peripheral portions of the centrifugal tract are endowed with similar properties. But it is of more interest to know what follows upon excitation of the sensory cell groups, which can easily be carried out in the spinal cord.

In this organ the grey motor column is surrounded with a thick layer of white matter, which prevents experimentation unless transverse section has been previously carried out. But the traumatism thus produced always brings about disturbances, among which is chiefly a great diminution of excitability of the deeper parts; hence we made use of the superficial position of the motor nuclei of the facial and hypoglossal nerves, in the experiments just described. Similar opportunities are afforded by the sensory grey columns of the cord, in the study of the excitability of the cells situated at the origin of the centripetal nerves; for the posterior horns are very superficial, and at the bottom of the posterior sulcus, nearly reach the surface, from which they are separated only by a very thin white layer in the intervals between the points of emergence of the sensory roots. The subjacent sensory cells can easily be excited by scraping this layer, or applying to it the electrodes.

This experimental condition is very favourable, since in all my experiments I always obtained results identical with those determined by excitation of the posterior roots themselves. I am speaking chiefly of results obtained on the horse, of which the cerebro-spinal axis lends itself well to these experiments, when the stimulus is applied after division of the cord from the encephalon, and at some distance from the point of section. I shall describe the case where, after division between the atlas and occipital bone (artificial respiration being carried on), excitation is made between the last dorsal and first lumbar root. On gradually increasing the current, we observe :—

(1.) Very weak excitations cause contraction strictly localised in the spinal and abdominal muscles near the point of application. The stimulus is then wholly reflected upon the cells giving origin of the motor roots corresponding to the sensitive roots near which it is applied.

(2.) Excitations with stronger currents are diffused in the cord, above and below the excited spot, but on the same side only. Unilateral and more extensive contractions of the trunk muscles appear, owing to reflexion of the stimulus into a larger number of motor roots.

(3.) On further increase of current strength, the contractions may spread not only beyond the trunk muscles, into the upper and lower extremity, the diaphragm and the neck, but also invade the opposite side of the body. The effects of the localised excitation are thus remarkably generalised ; the stimulus propagating itself to the ends of both lateral halves of the cord, and being reflected upon the motor cells along its passage on both sides.

Analogous results follow excitation of either of the two sensory roots between which the cord has been excited. These effects are exactly comparable with those following excitation of the sensory branch of the sternomastoid muscle.

The contractions are also localised in the muscles supplied by the nerve fibres, when the stimulus is weak ; they irradiate either on the same side, or on both sides, when it is strong.

Hence we conclude that the central motor and sensory cell-groups which give rise to the centrifugal and centripetal nerves respectively, behave on stimulation as these nerves themselves ; and that the theory of a nerve-circuit is thereby further justified.

I cannot present any experimental data as to the effect of excitation of the inter-cellular commissures, though I frequently attempted to obtain them. This excitation is possible only on transverse sections of the cord, and I have already stated that the resulting physiological disturbance vitiates all its effects. Generally speaking, superficial pricking or scraping with a needle, under these circumstances, remains without any effect, whatever part of the grey or white matter be touched. In order to obtain a result, we must suddenly plunge the needle to the depth of three or four mm.; and even then we obtain it only if we come across either the central ends of motor fibres or motor cells. There then may follow some localised contractions in the dorsal and lumbar regions; in any other situation these needle pricks have no effect.

This negative result is apparent when the sensory spinal tracts are alternately excited on the surface of section and on the natural surface of the organ. Scraping the latter at the level of the sulci and posterior columns, at some distance from the cut end of the cord, nearly always calls forth fine reflex contractions, which stand in contrast to the negative result of irritation of the divided surface of these posterior columns and cornua. This sensibility of the posterior spinal surface in mammals is observed only when circulation is going on regularly. If artificial respiration be stopped, and the animal be bled, death ensues, and this sensibility disappears very rapidly. It vanishes with the last heart-beat. We have seen that it is different in the case of the motor system, both motor cell-groups and peripheral nerves preserving their excitability for a few minutes after death. Hence every movement obtained by exciting the cerebro-spinal axis of a recently killed animal shows that the stimulus has excited some motor element. These indications are given for the benefit of those desirous of investigating experimentally the present subject.

(2.) *Interruption of the sensorimotor circuit in the cerebro-spinal axis.*—I shall not enter now upon pathological details calculated to clear up this point, but will confine myself to recall a few facts. Our main interest lies in the effects of destruction of the central sensory system, cells and fibres. Now we know that in tabes dorsalis, such a destruction, more or less extensive and complete, with preservation of the motor elements, produces locomotor disturbances which can be explained only as effects of central ruptures in the sensorimotor nerve-circuit of the muscles.

This is enough to demonstrate the function of this nerve-circuit ; and I shall not say any more on this subject, in the absence of new experimental data, to elicit which would be a difficult, if not an impossible, task.

V.—ON THE APTITUDE OF THE MUSCULAR NERVE-CIRCUIT OF ANIMAL LIFE TO CALL FORTH COÖRDINATED MOVEMENTS INDEPENDENTLY OF THE PSYCHO-PHYSIOLOGICAL CENTRES.

Towards the end of Part II., when discussing the mechanism of coördinated œsophageal contractions during deglutition, I said that this act depends on the continuity of the nerve-circuit, and on the activity of the central sensorimotor cell-groups from which the two arcs arise. This activity is such that the cell-groups must be considered as exciting centre and automatic regulator of the organs and process of deglutition. There are many more such in the medullary axis, presiding over sub-conscious motor functions, and the coördinating functions of which are admitted with reference to manifestations of vegetative life. But it is less generally recognised that this axis is similarly endowed with regard to movements of animal life, or locomotion [proper. A few appropriate examples will show the similarity between these two classes of functions, so far as the mode of action of the nerve-circuit, and its cellular centres, is concerned in both cases.

We shall consider two cases of experimental movements in horses after separation of the medulla from the encephalon. These contractions are generally considered only as muscular jerks in no way connected with movements adapted to an end. No doubt many reflex actions in the locomotor system appear to be such ; but in some we may easily trace distinct coördination.

(1.) *Artificial coördinated respiratory movements in horses after separation of the medulla from the encephalon.*—After atloido-occipital division of the cord, artificial respiration is carried out. When the latter process is suspended, whether after minutes or hours, there is never any restoration of respiratory movements; and asphyxia rapidly supervenes with hardly ever the least inspiratory jerk during the final convulsions. We can, however, under the circumstances excite reflex inspirations sufficient to keep up respiration, without the assistance of the bellows. It is enough to stimulate the perforating branches of the intercostal nerves. Horses very uniformly and readily react to this excitation, as I had very frequent opportunities of observing in the laboratory on animals prepared in the way just described. At the beginning of the experiment, before any weakening of the reflex powers of the cord, it is remarkable to see how the least excitation of one of the perforating branches sets up inspiration.

Any stimulus is efficacious; mechanical shock, squeezing, electric shock, tetanising currents. With a single strong excitation, we observe (along with some other movements which I leave aside) a short and ample inspiration. Rapidly repeated stimuli fix the chest in inspiration during their whole duration. The use of the sledge induction apparatus enables us to give to these respiratory movements the perfect thoracic rhythm with its increasing and decreasing characteristics. The electrodes being left applied to the nerve, the secondary coil is made to slide to and fro in a well regulated manner, so as to obtain the requisite augumentations and diminutions in the tetanising current strength. It is strange to witness how exactly this process can reproduce the normal respiratory movements, and how easily life is thus maintained after spinal section. By occasionally changing the nerves and the points of application I have often maintained artificial respiration for half-an-hour or even longer.

The coördination of the respiratory movements so obtained is perfect. They are not mere reflex jerks, but genuine contractions regulated towards the carrying out of a natural function. This coördinated, purposive character is even found in the short and sudden inspiration following a shock or instantaneous squeezing of the perforating nerve; such momentary stimuli acting upon the whole muscular respiratory apparatus. The chief muscle, the diaphragm, is thus thrown into activity not only in part or upon the excited side alone, but as a whole; and I have even seen subsidiary muscles, such as the external intercostals, contract at the same time.

These results show that the cord is capable of starting coördinated inspiratory movements, as for normal inspiration. This aptitude depends upon the cell groups belonging to the sensorimotor nerve circuit of the diaphragm, to mention only the essential muscle concerned. Under the influence of a special excitation these centres put forth their properties, along with those of their whole circuit, and the muscular function is thus carried on. In the present case the special excitation, which sets into activity the diaphragmatic nerve circuit, comes from a peripheral cutaneous nerve; under normal conditions it has a central origin: the medulla oblongata and the brain. Let us examine these two sources of excitation.

The bulb is the seat of what is correctly known as the respiratory centre, which sends off the rhythmical excitations necessary to keep up, during sleep and vigil, automatic respiration. After section of the cord, this centre cannot any longer act upon the cellular portion of the nerve-circuit of the inspiratory muscles; respiration then ceases suddenly, and cannot spontaneously recover itself unless the cells receive from elsewhere their needed rhythmical excitation—from the periphery, for instance, whence a stimulus could arise, more or less analogous to the experimental one through electrisation or squeezing the perforating intercostal branches. It is no doubt to such an intervention that reestablishment of respiratory movements after spinal division is due, as Brown-Sequard, and Wertheimer have observed it to be the case in dogs under certain conditions.

Let us now consider the case where the brain itself takes part in the respiratory function—when more than its automatic maintenance is involved. When respiration is to be slackened or accelerated, or made shorter or deeper, or used for special acts, such as vocalisation, coughing, sneezing, &c., we can understand that the higher centres enter into action to send specific excitations to the cell groups of the respiratory nerve circuits, either directly, or indirectly through the bulbar centre acting as a relay.

I only touch upon the surface of this difficult subject, avoiding all points not directly connected with this study. I hope thereby to present clearly the general ideas submitted to criticism on the respiratory nerve circuit, on the function of motor and coordinating centres fulfilled by the cell groups placed at the origin of this circuit ; finally on the relations between these spinal centres, and excito-regulating centres in the bulb and brain.

(2.) *Coördinated movements in the horse's hind limbs after medullary division.*—I come to the second class of facts, showing the capacity of the isolated cord to call forth coördinated purposive movements, through the influence of the central cell groups of the muscular nerve-circuit. The present case, though somewhat more complicated, may be briefly stated, because we shall have but to repeat ourselves when we come to state the deductions from the analysis of the facts.

For our present experiment a lively and nervous subject is to be selected (donkeys are very suitable), and prepared as before. If the medullary section has been well done, and the artificial respiration is properly carried out, the excitability of the cutaneous surface is well marked throughout, but especially about the hind limbs, where the least touch calls forth lively and more or less generalised reflex movements. If the pastern of the limb on the side opposite to that on which the animal is lying, be suddenly seized, the limb is suddenly flexed upon the trunk, with some alternatives of flexion, as if the animal consciously intended to withdraw it from the pressure. Sometimes, especially in the donkey, if the pressure be strong and almost instantaneous, this flexion is immediately succeeded by an equally sudden extension, as if the animal were kicking an aggressor. Squeezing of the collateral digital nerves still more readily excites this movement.

Thus in the two cases, peripheral excitation does not call forth disordered jerks, but associated and coördinated contractions, giving the impression of a locomotor act analogous to that produced by the same excitation in the normal animal. It is interesting to note that this reflex kick occurs chiefly among bad tempered subjects, naturally disposed to kicking as a mode of attack or self-defence. It is, therefore, not in lower vertebrata only that such spinal reflex movements having all the characteristics of perfectly coördinated actions, are to be elicited.

The mechanism of these locomotor manifestations also is readily explainable. The sensory peripheral stimulus calls forth the combined activity of the cell groups of the nerve-circuits supplying the limb, the segments of which perform regular movements, through contractions associated towards a true locomotor act. Here again the nerve-circuit of the muscles carries within itself the principle of motor coördination, when its central or cellular portion—a true locomotor centre—is brought into play by an excitation. After division the spinal locomotor centres can receive artificial peripheral excitations only, without which the muscles remain inert. When intact the cord receives impulses from the medulla, and the three large encephalic ganglia (psycho-physiological centres); in other words excitations come from all the central portions which rule automatic standing and walking, as well as all voluntary modifications of these actions.

Such are the facts I had to bring forward concerning the capacity of the nerve circuit of voluntary muscles, for calling forth coördinated movements independently of the higher centres. I have been led into saying a few words concerning the physiological relationship between these centres, and the spinal ones considered as origin of the two arcs of the circuit; my intention in so doing was merely to throw more light upon the mode of action of this circuit, without ever intending to enter upon the great question of the psycho-physiological centres, and their connection with the locomotor function.

VI. — HYPOTHETIC VIEWS CONCERNING THE INTIMATE MECHANISM OF THE NERVE-CIRCUIT OF MUSCLES.

No subject in physiology lends itself less to the determination of the intimate mechanism of phenomena, than the function of the nervous system. This is perhaps why we content ourselves with little, and cling tenaciously to that little. The current prejudices concerning the part played in muscular contraction by sensory motor nerve functions, are opposed to the adoption of the nerve-circuit theory. One is used to look upon the motor and sensory muscular systems as independent, and one is disinclined to admit that sensory nerves may have, in addition to their proper function, a direct share in the execution of movements governed by the motor system. One readily conceives an excitation starting from the anterior spinal cells, travelling down the nerve and the motor plates, and giving start to the contraction. It is then only that one calls in the centripetal system; the sensory terminations (?) take cognizance of the incipient contraction; this impression is conveyed upwards to the regulating centres, which in their turn reflect it upon the motor cells; and these, finally, react in consequence, that is by regulating the contraction whose apparition they have already determined. I attribute greater simplicity to this mechanism, by admitting absolute simultaneity of collaboration among both motor and sensory nerves.

In order to frame a sufficient hypothesis concerning the synergy of both kinds of nerves in coördinated motion, we must keep in view the nature of the physiological relationship between both arcs at their extremities, where we assume them to be anatomically connected together. This relationship is such that an excitation is easily carried from the centripetal to the centrifugal conductor, through the central loop of the circuit, whilst no such transmission takes place through the peripheral loop, from the centrifugal to the centripetal conductor. Thus, this peripheral loop, or the muscular tissue which is tangent to it, may be considered as an insuperable barrier for excitations conveyed by the circuit; applied to the peripheral end of the centripetal conductor, an excitation can reach the muscle only after travelling through the whole circuit, first upwards to the central, then downwards to the peripheral, loop. This is the fundamental fact to be remembered. Thus an excitation runs round the circuit in one direction only. Still there is reason to believe that the disturbance it sets up on entering the circuit is felt in both directions, for it is difficult to understand this disturbance to be other than a vibratory motion, giving rise to systems of waves which are propagated towards both extremities of the conductor. Some waves are *præ*pulsive, going in the same direction as the excitation; others *retro*pulsive, going in the opposite direction. This mechanism presents a certain analogy with what takes place in an air-filled tube, in which the same stroke of a piston produces in front a condensed half wave, behind a dilated half wave. Each of these is propagated in opposite direction to one extremity of the tube, as we supposed the case to be with reference to the præpulsive and retropulsive nervous waves.

In order better to define the action of this mechanism, let us consider the physiological event in which it has taken part, that is when an excitation impinges upon the nerve circuit in its central part. We may suppose that this excitation—whatever be its origin, brain, bulb, cutaneous nerves—first reaches that part of the circuit which stands between the centripetal and centrifugal cells. In the latter the excitation sets up a præpulsive wave forwards; in the former a retropulsive one backwards. Both these waves reach simultaneously the peripheral loop and exert their influence upon the muscular tissue that lies tangent to it; this influence is chiefly motor in the case of the præpulsive, chiefly coördinating in that of the retropulsive wave. For we have seen that if this simultaneous arrival of the two waves is not necessary to the provocation of muscular movement, it is essential to its coördination. Interruption of the centripetal conductor, by preventing the arrival of the retropulsive wave, does not necessarily paralyse the muscle, but it always interferes in some measure with the physiological adaptation of muscular contraction to the execution of true coördinated movements. If one cannot foresee the essential cause of this disturbance, one has at least a clear notion that the lesion which produces it disables the centripetal conductors from transmitting to the muscle, under the shape of the retropulsive wave, the effect of the excitation experienced by the central portion of the nerve-circuit. We need say no more on this subject.

I might now review all cases of interruption or artificial excitation of the several parts of the nerve circuit, and show how the present theory adapts itself to our experimental results. But this would be superfluous. A general indication only need be given, concerning artificial excitation. Whatever be its point of application, it always calls forth a contraction if its præpulsive wave can reach the peripheral loop, and through it the tangent muscular fasciculus. The retropulsive wave has no share in the result, and to it is indifferent whether the centripetal branch of the circuit be or be not broken. For instance, if the motor nerve be divided and excited close to its termination the præpulsive wave will produce contraction just as well as if a retropulsive wave had circulated backwards round the whole circuit. Again, if after division of the sensory nerve close to its termination, the central aid be excited, a præpulsive wave only is set up, sufficient to produce contraction. On the other hand, a retropulsive wave alone, set up by excitation of the central end of the centrifugal or the peripheral end of the centripetal conductor, never gives rise to any muscular contraction.

To sum up, the retropulsive wave intervenes only in the mechanism of coördinated movements, provoked by natural excitation of the spinal cells of the nerve-circuit.

We might institute a comparison between this physiological work, and the corresponding electric modifications it sets up in the nerves; but I must confine myself to the statement that the study of the negative variation yields inductions favourable to the theory of præpulsive and retropulsive waves, and so does that of the electrotonic state above and below the excited spot.

Such are the considerations which I had to present on a possible manner of understanding the mode of action of the muscular nerve-circuit, that is to say the intimate mechanism of the cooperation of the muscular sensory fibres with the motor in the execution of movements.

Are these two kinds of fibres really so joined at their extremity as to form a circuit in which excitations give rise to waves in both directions? I may not affirm it; "sunt verba" may it be said of all what precedes concerning the possibility of such a mechanism. But this cannot be said of the facts which this attempt at synthesis is intended to bind together. Whatever fate be reserved for the theory herein exposed, interesting results will remain from the experiments undertaken to verify the theory of a muscular nerve circuit.

Addendum.—Among the new facts published in this paper, I may mention:

(1.) All results from experiments on the sternomastoid muscle; excitations and divisions of its motor and sensory nerves.

(2.) All experimental facts related to excitation and division of centripetal nerves of lower cervical œsophagus.

Some other facts had already been very incompletely mentioned in my previous publications, especially

(1.) Physiological respiration kept up by rhythmical excitation of perforating intercostal branches in the horse after spinal section.

(2.) Coördinated movements of the hind leg under similar conditions set up by excitation of the cutaneous nerves.

I do not believe I ever published before a large series of experiments made on the complete sensory enervation of one foot in the pigeon, and its effect upon standing and walking. But the facts elicited thereby have now lost the interest of novelty.

(Translated, from the original MS., by the Editor.)

THE SENSE OF EFFORT: AN OBJECTIVE STUDY.[1]

BY AUGUSTUS D. WALLER, M.D.

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§ 16. Hypothetical considerations and general conclusions.

§ 1—Introduction.

As is well known, the opinion very generally referred to as Bain's, but previously taught by Johannes Müller and by Ludwig, and still held by Wundt, Hughlings Jackson, Crichton Browne, and many others, to the effect that the sense of effort is a subjective concomitant of the outgoing nerve flow, is not in fashion with most working neurologists of the present day.

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[1] The substance of this paper was communicated to the Neurological Society (St. Mary's Hospital, May 21st, 1891) partly in printed abstract, partly by description and by demonstration of methods.

The report of the debate on the "muscular sense," held four years ago, between Drs. Bastian and Ferrier, contains most of the preliminary considerations which it would otherwise be proper to allude to in introduction of the present matter. Many of the considerations now offered are in fact a direct outcome of that debate, at least in one fundamental branch of it. (v. "BRAIN," April, 1887.)

The argumentative character of §4 is in consequence of communications to and from Dr. Bastian, who, as is well known, is the most prominent upholder in this country of muscular government by kinæsthesis.

Portions of the subject considered in §§9, 13 and 16 are of necessity alluded to in this connection, but will be more fully described at a future opportunity.

The paper is subdivided into paragraphs in order to facilitate judicious "skipping" by either physiological or non-physiological readers.

Bastian goes the length of referring to it as "a doctrine now disproved,"[1] and Ferrier "is thoroughly in agreement with Bastian as to the sense of movement being dependent on centripetal impressions and not on outgoing currents."[2]

There is however, as Dr. Jackson says,[3] no reason why states of consciousness should not attend activities of motor as well as activities of sensory cells.[4] And Sir J. C. Browne,[5] in the question and argument which he contributed to the discussion on the muscular sense held four years ago, very aptly illustrated the *a priori* tenability of the proposition. In fact, as it seems to me, the proposition and its negative are of equal weight, equally admissible as statements of opinion, equally unproven and undisproven by the arguments invoked for and against them. *A priori* tenability is not proof, but failure of proof is not disproof. And I cannot admit as a positive premise either the opinion of Bain, nor the dictum of Bastian—the first to the effect that we know our movements by a central sense of motor emission, the second to the effect that the sense of effort as a concomitant of central motor innervation, has been disproved. An appeal to our own feelings of effort does not prove one thing or the other; observations of clinical phenomena leave the matter unsettled; experiments on animals give answers which must be dressed out with interpretation and inference.

There remains, however, in my opinion, very definite and accessible *objective* evidence to be obtained by the study of the manifestations of fatigue on man, quite apart from introspective self-analysis. There are also definite physiological data, derived from experiments on animals, which can be utilised in simplification of the problem upon man, data which, it appears to me, have not yet been sufficiently taken into reckoning by even the most precisely analytic of our neurological leaders and teachers.

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[1] "The Muscular Sense" BRAIN, April, 1887, p. 120.

[2] *Ibid*, p. 89.

[3] *Ibid*, p. 107.

[4] Of course the distinction implied between "motor" and "sensory" in this short quotation is not Dr. Jackson's, but the meaning of the words is not here in question.

[5] "*Ibid*, p. 103.

§ 2—Exclusion of Nerve-Fibres.

I am referring more particularly to the now clearly ascertained fact that nerve-fibre is practically inexhaustible by nervous action.[1] Bernstein's experiment (1877) supplied evidence that nerve is much more resistant to excitation than muscle. The experiments given in my reports to the British Medical Association (1885 and 1886) brought evidence to show that nerve continues to be excitable long after its excitation fails to act on muscle, which itself is still directly excitable. In other words, that the

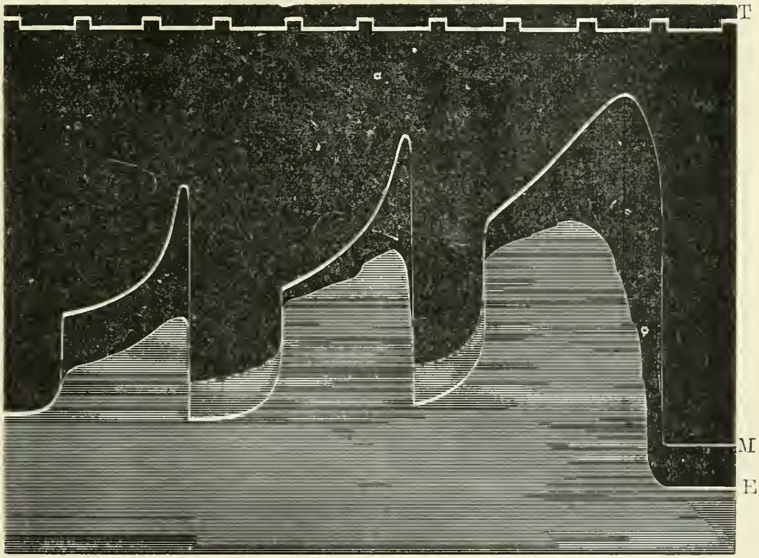

FIG. 1.

Simultaneous tracing of three muscular tetani and of their accompanying electrical effects taken upon frog's gastrocnemius by means of a capillary electrometer, to shew the comparatively rapid fatigue decline of the mechanical effect (line M), and of the electrical effect (line E). The time line T indicates minutes.

N.B.—ALL TRACINGS IN THIS PAPER READ FROM RIGHT TO LEFT.

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[1] In proof of the fact that this non-exhaustibility of nerve fibre is not generally recognised, I may perhaps quote the following passage from Hughlings Jackson's Lumleian lectures on convulsive seizures (1890), in which the experimental exhaustibility of nerve-fibre is alluded to and used as a well-established term of comparison.

"The nervous elements exhausted in post-epileptiform paralysis are, I motor end plate is the weak link in the neuro-muscular element. Wedenskii's observations, published the year before (1884) brought evidence to show that nerve is indefinitely excitable. The electrical tokens of excitability were the test utilised in both cases. Wedenskii used the telephone to detect the negative variation of faradised nerve,

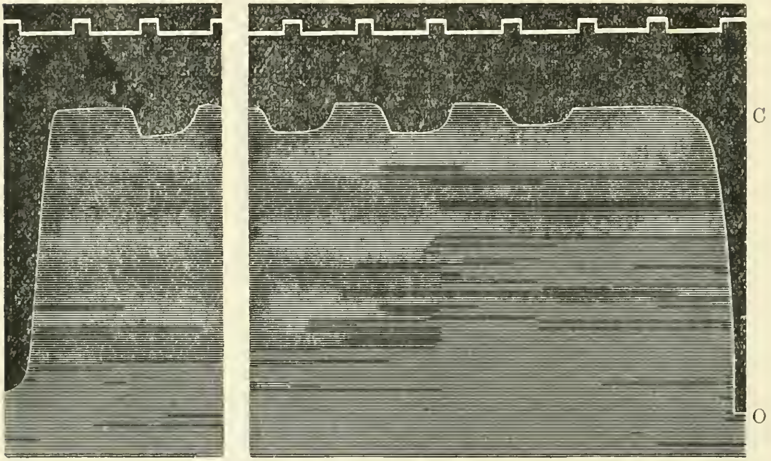

FIG. 2.

Tracing of the electrical effects of nerve tetanisation taken upon a frog's sciatic, to shew the absence of fatigue decline. Each period of tetanisation lasts about one minute, and is separated from the next period by an interval of rest, also about one minute. Only the beginning and end of the original tracing are given; the first portion includes three periods of negative variation, the last portion only one, and six intervening variations are omitted. The zero level is indicated at O, the injury level is at C, each negative variation is formed by a depression from C towards O. It is to be noticed that the last variation is as pronounced as the earlier ones, *i.e.* there is no evidence of fatigue.

suppose, in the same state as are the motor fibres of the sciatic nerve going to the cut-off leg of a frog after strong faradisation of that nerve-trunk; the leg is convulsed by the faradisation (stage analogous to the epileptiform seizure), and is next paralysed (stage analogous to the epileptiform paralysis) because its motor-fibres are exhausted by the unnaturally high functioning they have been artificially compelled to. A closer analogy may be stated, although the case to be instanced is a complicated one. The motor nerves of a frog, poisoned by a very large dose of strychnine, lose function partly by the direct action upon them of that poison, but partly (this is what is relevant) through exhaustion—the exhaustion of over-use, due to the intense activity of the nerve during the stage of spasm."—*British Medical Journal*, April 12, 1890, p. 821. The last line is quoted from Wood's Therapeutics, 7th edition, pp. 258-9.

to which method it may, however, be objected that no distinction is thus possible between the negative variation and electrotonic currents. I used the galvanometer, and was careful in later experiments to distinguish between a true negative variation and electrotonic currents. Finally, Bowditch (1885 and 1890) has demonstrated the indefinite endurance of nerve on mammalia by the curare method, in which there is, of course, no fear of the electrotonic fallacy. His experiment is shortly to the following effect:—An animal (cat or dog) is curarised, faradisation is kept applied to the sciatic nerve for hours, and when the curare effect wears off, muscular contractions make their appearance, proving that the nerve itself retains excitability after faradisation for three, four or five hours.

This is one of the objective items which, apart from its intrinsic interest, must be considered as contributory to an accurate notion of the incidence of the feelings of fatigue, effort, and movement. We may, in fact, at the very outset exclude alterations of nerve from any considerable share in the production of such feelings; nerve, in so far as it is accessible to physical examination, is pre-eminently a passive and force-transmitting organ, and not an active force-producing, or rather force-transforming organ. It is, then, not to be wondered at that neither chemical nor thermal changes have ever been demonstrated in nerve however long, or however strongly, it may have been excited; the only evidence that excited nerve suffers material change is supplied by the electrical signs of activity. This last item of evidence forbids us to assert absolutely and theoretically that nerve-fibre is unaltered by excitation; all we may properly infer from experiment is that fatigue of nerve-fibre is practically a zero quantity.

§ 3—The Distribution of Fatigue Effects.

The exclusion of nerve-fibre is only a preliminary and negative item. The positive item or series of items, which I now desire to submit to criticism, is constituted by an attempt to determine what share the more important and active elements in the cerebro-muscular chain—cerebral cell, spinal cell, end plate, muscle fibre—take in the expenditure of energy and its resultant "fatigue."

I shall most quickly get into the matter by quoting the conclusions contained in a summary report made some years ago to the British Medical Association, and from this point of departure by demonstrating the methods and instruments followed in the prosecution of the enquiry, and by stating in what particulars I am able to add to and harden those conclusions, or obliged to make withdrawal from or reservation to them.

"Mammalian muscle in its normal relations, as regards circulation and nutrition, may be almost indefinitely subjected to single break induction shocks at intervals of one second; and the record will show that the extent and duration of contraction is not less at the end than at the outset of the experiment, nor is there any sensation of fatigue.

"The endurance of normal muscle is such as to constitute a strong testimony in favour of the view, that the sensation consequent upon prolonged muscular action, to which we give the name of "fatigue," is normally central and not peripheral as to its seat; or, at least, that fatigue commencing first in the central nervous system is protective from peripheral fatigue. The explanation of this great resistance of normal muscle to fatigue is quite obvious; it depends upon the great rapidity with which the muscle is restored during repose; and normally this restoration is such, that single induction-shocks remain without appreciable effect, however prolonged. To obtain any distinct evidence of fatigue in normal muscles, they must be subjected to the continued action of tetanising currents; under these circumstances, if an interruption frequency be chosen, such as to give a tetanus not quite complete, but still showing the serrated top indicative of the contractions which compose it, it will be seen that the serration gradually becomes lost, and the tetanus completed if the excitation be sufficiently prolonged. The fact (which I have had occasion to verify) has already been observed by Marey, and need therefore only be alluded to here as being a phenomenon of the same nature as the well-known gradual fusion observed on frog's muscles, and which may, therefore, be regarded as a sign of fatigue.

"This view, that fatigue consequent upon prolonged muscular exercise is normally central rather than peripheral, receives further support from experiments with the dynamometer, which I arranged as a dynamograph, in order to obtain the record of a succession of greatest possible voluntary efforts of muscles of the forearm; the duration of contraction and that of rest being regulated by means of a metronome. Such series are characterised by a comparatively rapid and surprisingly regular diminution, contrasting markedly with series of tetani provoked by peripheral stimulation; and the single contraction, provoked by a break induction-shock, at a time when the muscles have become incapable of further voluntary action, nowise differs from a contraction taken at the outset of experiment.

"The beneficial effect of 'massage' upon fatigued muscles is not in harmony with the view expressed above, and I do not commit myself to a denial of all peripheral share in 'fatigue,' but only to the assertion that fatigue is normally of central origin, and protective from peripheral fatigue."

Of the work which has been done more recently on this subject the most considerable is that of Mosso, and of Maggiora, and of Lombard working under Mosso's directions.

Mosso's observations were taken by the isotonic method (which is the method with which we are most familiar in the laboratory as that by which the laws of fatigue in excised frog's muscle have been studied by Kronecker, Tiegel and their successors); the instrument employed for the purpose, termed by him the ergograph, essentially consists in a weight attached to a recording slider to which the middle finger of the otherwise immobilised hand and forearm is attached. The great advantage of the method consists in the fact that the muscular effect is measurable in terms of work, in kilogrammetres—the weight being known and the height to which it is raised recorded. I will quote some of Mosso's principal experiments and conclusions simply to show the kind of indications afforded by this method, with the flexor muscles of the middle finger. Like Fick, he finds that artificial contractions can only raise a much smaller weight than voluntary contraction. He shows that after voluntary action has ceased in the muscle, direct excitation can evoke fresh work; and *vice versâ*, that after direct excitation has ceased to effect any work, voluntary action can do so. He finds that intellectual work reduces the muscular power of work, and the same fact is even more strikingly demonstrated by Maggiora's experiments. The latter observer, moreover, shows that loss of sleep and fasting make muscle more fatiguable, while food and massage have an almost immediate restorative effect.

Mosso's conclusion with regard to the question immediately before us is "*Die Abnahme der Leistungsfähigkeit bei der Ermüdung hängt nicht nur von musculären Veränderungen ab. Es ist schon bekannt, das wír die Anstrengung von der Arbeit unterscheiden müssen : und ich glaube, dass bei jeder Anstrengung noch zwei Theile zu unterscheiden sind —die centrale oder Nervenanstrengung—und die peripherische oder Muskelanstrengung;* and although it is not easy to form an estimate of the proportion between the two factors, a comparison between Mosso's data (in which work is the measure of energy) and my own (in which absolute force is the measure of energy) shows that in the identical conclusion we have both arrived at, more stress is laid on the peripheral factor by Mosso, whereas I have laid more stress upon the central factor. But the methods are different, and it is not clear whether work or force is the more faithful index of neuro-muscular energy. Fick has of late insisted upon the isometric method as yielding a more practicable and correct indication of muscular contraction than the isotonic method under the complex and varying mechanical conditions of muscles, bones and joints of the human subject.

Mosso's method, by weight to be raised, exhibits how much work a given muscle can do under the conditions of experiment, but the maximum at the beginning of a fatigue series does not necessarily show the maximum work obtainable, nor does the zero elevation at the end of a series really signify that no more work can be done. On the other hand, Fick's method, by spring slightly deflected, exhibits the absolute strength of muscle undergoing a negligible diminution of length in its contraction; the maximum effect at the beginning of a fatigue series is a true measure of maximum energy produced, and the effect at the end of the series never reaches zero, but indicates to what degree the "workability" (*leistungsfähigkeit*) has declined.[1]

§ 4—Nature and Scope of the Inference.

It will probably be admitted that the sense of fatigue, consequent upon prolonged or repeated effort must be regarded as similar in kind to the sense of effort experienced when we put our full fresh strength into action, and that the latter again is similar in kind to the muscular sense considered to be the concomitant of all muscular contraction. The sense of movement, the sense of effort, and the sense of fatigue are degrees of the same sensory phenomenon, or rather this similarity is the basis of the experimental proofs I have to offer. If a distinguishing line is to be drawn between sense of maximum effort in a fatigued state, and sense of maximum effort in a fresh state, or between sense of maximum effort and sense of moderate effort, then my proofs must be rejected, for they depend upon measurements of the declining maximum effort in fatigue and determination of the seat of loss of energy upon which that decline depends.

Therefore I put this as a definite question: Is the relationship admitted or denied, and if denied, why so?

The question just put is fundamental—the entire case as I am now offering it is made to turn upon it—the very title which I have chosen contains by implication an affirmative answer. I therefore felt bound not to shirk the point, but to bring it up prominently for examination.

Dr. Bastian, as was to be expected, refuses to admit the assimilation of these three items under one genus; he says (in a private communication), that—

"What is commonly meant by the 'sense of effort' seems to me to be something wholly different from the matter you have been investigating. I cannot at all admit that the impressions which we receive as accompaniments or sequences of movements have anything in common with the cumulative general feelings which we obtain as a result of some continuity of neuro-muscular action.

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[1] The designations "isotonic" and "isometric" were introduced by Fick. It should be mentioned that in the isotonic method, the shortening of muscle extended by a small and constant weight is measured, whereas in the isometric method the muscle is almost completely prevented from shortening by a strong spring, the minute deflection of which is magnified by a very long lever. The first-named method demonstrates kinetic energy, the second potential energy. In the frequent references which will have to be made to these methods I shall refer to the first as the *weight method*, to the second as the *spring method*, and to a third method, by which the lateral effect accompanying muscular contractions are recorded, as the *bag method*.

Now this is a denial *in toto* of the position I have taken, but it is a simple denial without reason given. On the other hand I have made a simple assertion, also without reason given. To make good that position reasons must be forthcoming; these must be of greater weight than any reasons which may be forthcoming for the denial. To demolish the position good reasons must be forthcoming for the denial, and these must be of greater weight than any reasons which may be forthcoming for the assertion. Failing one or other of these two alternatives neither assertion nor denial can be admitted as established. I shall therefore give the reasons for my assumption at some length, with perhaps some tedious iteration to the following effect:—

The sense of effort and the sense of fatigue are not "senses" properly speaking; they are sensations. The first is a sensation accompanying muscular action; the second is a sensation consequent upon muscular action. The first owes its being to molecular changes which accompany muscular action; the second to molecular changes which have accompanied muscular action, *i.e.*, they have a common cause, the changes which produce the first also produce the second, and it is not material to our argument whether the positive changes with the effect are succeeded by positive or by negative after-changes, as the substratum of the after-effect. The subjective difference between the two sensations is absolutely no evidence against the necessary and fundamental fact that they have a common material cause. The first is the positive effect, the second is the positive or negative after-effect, and the molecular changes upon which they depend occur in the same situation. They are comparable with the image and after-image in vision which are the positive effect and the negative or positive after-effects of molecular changes in a given retino-cerebral area. Would Dr. Bastian argue that because yellow appears different from blue, yellow and blue have nothing in common; or that to determine the physiological locus of a blue after-effect would not be to give evidence of the physiological locus of a yellow effect?

Now my contention is this: we have no means of determining the incidence of the material changes underlying the motor effect; we have means of determining the incidence of the material changes underlying the motor after-effect. The seat of both is identical. If we determine the one we also determine the other.

The sense of effort (taking the expression to include the muscular sense) and the sense of fatigue, are sensations—the first *with* action, the second *after* action.[1]) *Id est*, both the sensory phenomena are attributable to the same cause, and we may expect to find the effect "shadowed" or "mirrored" in the after-effect. If we find in the cerebro-muscular chain objective signs of the after-effect more and less pronounced at the different stations of the cerebro-muscular chain, we must infer that the effect has been similarly more or less pronounced in the same situations.

I am most fully conscious that the point of departure and direction of thought are the main determining factor of the end of a train of thought; I submit that to make the sensory phenomenon the point of departure has not led to assurance, that the investigation of sensory alterations is full of pitfalls, and that to take the demonstrable material alterations of matter accompanying *and following* its activity, as our point of mental departure, can give positive information.

Let me illustrate the real and possible problem by comparing it with an imaginary and impossible problem.

By looking at a white patch, the retino-cerebral apparatus is excited—suffers material change. It is asked whether that change is in the retina or in the cortex, and no answer other than conjectural is forthcoming. But a grey after-image of the patch follows—being the negative after-effect, and necessarily attributable to material change in the same parts. If the incidence and repartition in retina and in cortex of the physical changes producing the after-effect could be determined, it would be legitimate to infer that the physical changes producing the effect had a similar distribution. Turn to the analogous *possible* case. We make a movement and may have a sensation, attributable to material changes somewhere or everywhere in the cerebro-muscular apparatus. We cannot determine their repartition, but we make many movements and then investigate their material legacy, and by objective signs determine its repartition in the chain. Must it not be admitted from the material standpoint that the impalpable changes accompanying muscular action are summed and mirrored (or shadowed) in the palpable changes following such action, and that in the determination of the repartition of the after-effect in centre and at periphery, we have an indicative ratio to the repartition of the effect?

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[1] It is not essential to the argument whether we consider a fatigue change as a positive or negative consequence of an effort change. Comparisons between the two sensations are of no value to decide this point; it is a choice between probabilities, which, without entering upon argument, may be simply stated; it is probable that an effort sensation is the concomitant of a disintegrative change; it is possible that a fatigue sensation is the concomitant of a summated disintegrative change (positive after-effect), but it is also possible that it may be the concomitant of an integrative process (negative after-effect).

The affirmative answer to this position is the major premise of this paper; the study of fatigue-phenomena is its minor premise; and we may now clearly see and formulate our task as follows:—

Major Premise.—*The material changes, subsequent to action, are co-extensive in the motor organ with the material changes accompanying action.*

This proposition is simply a statement that effects have positive or negative after-effects, as illustrated by retinal phenomena. Or otherwise that an effect can be investigated in its legacy, which is its shadow or its reflection.

Minor Premise.—*An investigation of the distribution of fatigue effects*, i.e., *action after-effects, in the central and peripheral components of the motor organ.*

Conclusion.—*Propositions asserting the distribution of action effects in the motor organ which conceivably underlie the accompanying sensations inaccessible to objective study.*

The only possible objection left to this argument (as far as I can see) is that the subjective sensation and the objective motion are assumed to be produced by the same alteration of the same substance. It may be objected that the sensory effect is caused by the alteration of a different substance from that which is altered in kinesis—that the kinæsthetic effect is not due to alteration of the inogenic substance either at the centre or at the periphery, but to alteration of an æsthesogenic substance. This, although a strange argument to be used by anyone convinced of the muscular origin of the sense of effort, must nevertheless be considered. To this it may be replied—

(1) That material, other than inogenic,[1] contributes to the sensation of movement, is not denied; the part played by the peripheral passive elements subjected to movement, *i.e.*, the skin, the articulations and the connective tissues, is admitted.

(2) The parallelism between the sense of effort and the kinetic effect, and between the sense of fatigue and the loss of kinetic power, indicate that even if the subjective and objective effects be referred to two substances, the varations of the latter, as shown by its power, must be admitted as indicative of variations of the former.

But it may further be objected that even assuming either one substance or two, the subjective effect may be due to an alteration at one part more than another of the motor organ, or at one part exclusively, *i.e.*, in the centre alone or in the periphery alone. And this, in fact, is practically the position taken up by Dr. Bastian, who locates the kinæsthetic stimulus exclusively at the peripheral end of the motor organ. This position is incontrovertible by objective proof; the *ultima ratio* is the personal ratio of the thinker, which will determine his choice between a conclusion pointed to by demonstrable kinetic variations at the different points of the cerebro-muscular chain, and an undemonstrable kinæsthesogenesis limited to its peripheral end. I do not know of any verbal analysis of

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[1] "Inogen," "inogenic material," are terms introduced by Hermann, and are convenient to denote the hypothetical force—producing material to the change of which muscular movement is attributable. As used above the term denotes changes in the whole cerebro-muscular chain.

Bastian's kinæsthesis, or of any objective analysis of neuromuscular phenomena, which can rescue the word from its purely subjective and purely imaginative connotations.

Admitting as true the major premise, admitting as highly probable that the kinetic signs of material changes in the motor organ are the same as, or similar to, or parallel with, the material substrata of the concomitant sensificaatory phenomena, I proceeded to the experimental determination of the incidence of normal voluntary fatigue as the minor premise, in the belief that an answer to this specific question would carry with it the true answer to the more general question as to the place where molecular change occurs in normal voluntary action, and the highly probable answer to the still more general question as to the origin of kinæsthetic phenomena.

If, however, anyone still denies the common causation and parallel distribution of the effect and after-effect, or admits minor premises derived from other sources as outweighing the data upon which I rely, then my results *qua* the after-phenomenon, called "fatigue," are limited to that special state, and do not carry the physiological answer which I claim they do, as regards the incidence of the phenomenon indicated by the various expressions, "sense of effort," "muscular sense," "sense of movement," "sense of innervation." But it is incumbent upon such a person (1) to deny and controvert the major proposition; or (2) to show errors of observation or insulation, in the experiments of which the minor propositions are the expression; or (3) to adduce positive counter-data outweighing my experimental data. A critic successfully taking the first or third course would destroy the conclusion, leaving the facts intact *qua* fatigue; by the second he would demolish facts and conclusion together.

§ 5—The Experimental Question Stated.

The question is: does normal voluntary fatigue depend upon central expenditure of energy, or upon peripheral expenditure of energy, or upon both factors conjointly, and if upon both, in what proportion upon each?

The form of this question is justified as follows :—

A maximum voluntary effort may decline,

(1) By decline of cerebral motility. }
(2) By sub-cerebral or spinal block. } *Central.*
(3) By motor end-plate block. }
(4) By decline of muscular energy. } *Peripheral.*

On the human subject it is impossible to separate factors 1 and 2, we must therefore embrace them under the term central.[1]

The third factor can be more easily demonstrated upon isolated parts of lower animals ; its existence can also—if less assuredly—be demonstrated upon man ; but for the clearer appreciation of the central *versus* the peripheral factors I do not dwell upon this sub-distinction, and include the effects of factors 3 and 4 under the common term peripheral.

May there, in addition to these four factors, exist other and disturbing factors ? Does fatigue of nerve-fibres contribute to the decline of a voluntary action ? May subjective factors arising from sensations of peripheral origin come into play and falsify the objective signs of motor fatigue ? The first of these questions has been answered above : nerve-fibre fatigue is a zero or an infinitesimal quantity ; to the second question the answer is that subjective sensory factors can be eliminated by proper adjustment of the conditions of experiment.

§ 6—The Experimental Answer and the Conclusions to be Inferred from it.

It may seem a very simple matter to institute a crucial experiment, giving an answer to the enquiry whether fatigue is manifested at the centre, at the periphery, or at both, and if at both, in what proportion at each. In reality, it is practically not at all easy, and considerations of method very soon press themselves upon our attention.

By means of the dynamograph (*vide infra*) I took series of maximum voluntary efforts at regular intervals during regular periods, viz., efforts lasting two seconds, separated by intervals of two seconds; efforts lasting two seconds, separated by intervals of one second; efforts lasting two seconds, separated by intervals of four seconds. I also took groups of such efforts, separating each group from its successor by a period of repose. The object of these observations was to form a preliminary estimate of the rate of decline of energy, and the rate of recovery, without however any attempt to say *where* in the cerebro-muscular apparatus the alterations of energy take place. Leaving on one side the results of this inquiry, I pass on to the question which is now before us, viz., the locality of the fatigue effect. I first attempted to compare a series of maximum voluntary contractions of this kind with a series of maximum direct tetani of equal duration at equal intervals provoked by direct faradisation of muscle and of nerve, in the anticipation that a comparison of the decline in the two cases might furnish data towards this conclusion. The observation in this form proved to be unsatisfactory. Using the hand and forearm as the tested object, I could obtain no dynamographic tracings by the direct excitation of muscle and nerve at all comparable with those of voluntary efforts. The grasp of the hand was very ineffective, partly, no doubt, because extensors as well as flexors were put into full action; but partly also, as is now known from Fick's observations, in connection with the fact that maximum *voluntary* contraction of a muscle is far more powerful than maximum faradic contraction. I was, therefore, obliged to give up my original plan, and to adopt a different system of comparison. Using, as before, the dynamograph, I added below the record of the grasp of the hand, that of the hardening of the muscles of the forearm, using for the purpose an elastic bag fixed to the arm by a leather bracelet, and connected with a Marey's tambour.

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[1] I have given experimental evidence to show that on the lower animals the two parts in the cerebro-spinal organ do not fail together but successively. (*British Medical Journal*, 1886, p. 102.)

Before proceeding further it was necessary to compare the indications thus obtained with those of the dynamograph in order to ascertain whether the former is at all capable of yielding a measure of the muscular energy under various conditions. This was a simple enough matter, and led to some significant results. The ordinates or excursions of the lever caused by muscular hardening, although not exactly proportional with the ordinates or excursions of the dynamograph, do, nevertheless, under certain conditions, vary up and down in a sufficiently indicative manner—are in fact very nearly proportional. This having been ascertained, a bag is strapped to the forearm, maximal faradisation is applied, the hardening is recorded. A series of maximum voluntary efforts are made, and at its end without any delay, maximum faradisation is again applied and the hardening recorded. *Thus recorded there is no apparent diminution in the direct indications after a voluntary series of efforts during which the energy fell* 50 *per* 100.

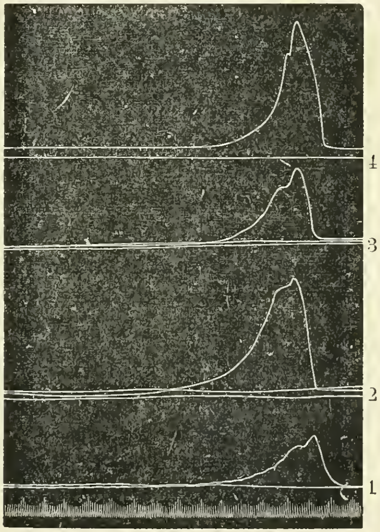

FIG. 3.

Four single contractions of the muscles of the fore-arm, by break induction shocks of uniform strength, recorded by means of a bag strapped to the limb.

1. Normal. 2. Immediately after voluntary fatigue. 3. After a period of repose. 4. Immediately after renewed voluntary fatigue.

The time line indicates $\frac{1}{100}$ths of a second.

NOTE.—This statement, as well as those already quoted above, are apparently contradictory of other data which will be described. Nevertheless the statements are left as written, and occasion is taken to call attention to an important point of method which will be more fully considered and utilised further on. The statements referred to apply only to lateral or bag indications. We shall find that although there is a general parallelism between the bag and the spring indications in the normal state, so that both vary up and down together, this parallelism is departed from in the fatigued state, the lateral or rigor effects being much less diminished than the longitudinal or dynamic effects, and the spring being a far more sensitive indicator of fatigue than the bag. It may even happen that single contractions recorded by the lateral method are actually greater in fatigue than in the normal state, as is illustrated in Fig. 3. This point having been recognised we shall systematically take double records of the lateral and of the longtitudinal effects simultaneously.

Maximum effects grouped in two or more series give very significant results. If we take two fatigue series of maximum endurable direct effects, separated by a fatigue series of voluntary maxima, we shall find that, so far from causing a further reduction of direct capability, the voluntary series has not prevented a recovery of power *which can only have taken place in peripheral organs.* This recovery seems all the more surprising when we reflect that it must be due to a reintegration in a tissue which is kept under a disintegrative influence. The motor end plate suggests itself as the weak link at which a temporary protection is set up, allowing reintegration to overtake and surpass disintegration.

Or, varying the experiment, if we take two fatigue series of maximum voluntary efforts separated by a period of maximum endurable direct faradisation, we shall see obviously enough on examination of the records, that the direct excitation, so far from causing a further reduction of voluntary capability, has not prevented a recovery of power *which can only have taken place in central organs.* I cannot see any escape from this conclusion; it may perhaps be urged that direct faradisation is less effectual and therefore less "fatiguing" than volitional tetanisation, but to this the rejoinder would be that we have witnessed evidence of *recovery* somewhere in the cerebro-muscular chain *during* maximum bearable direct excitation and therefore in spite of local

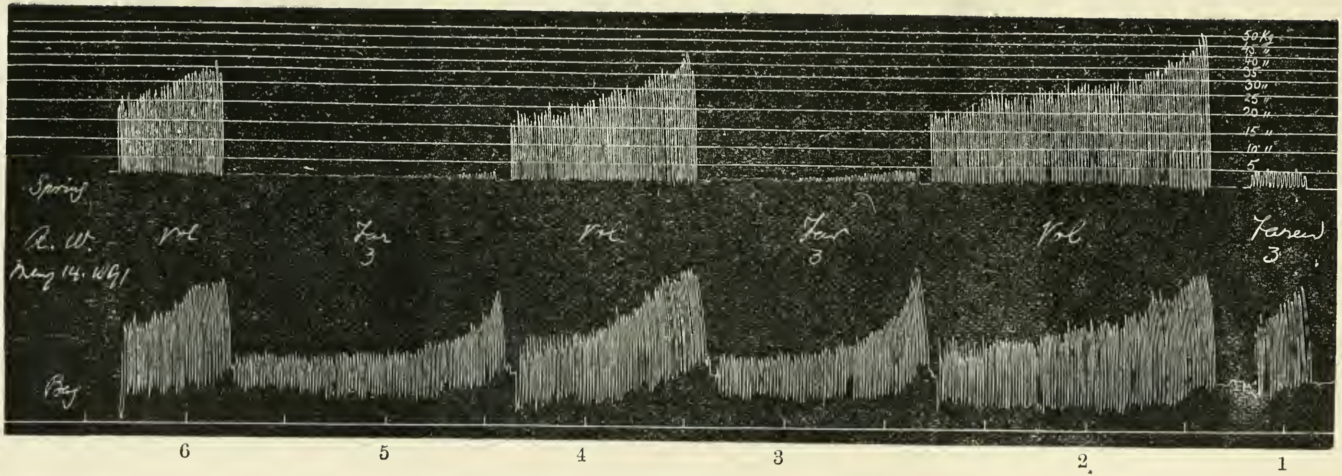

FIG. 4.

Groups of maximal, voluntary and direct muscular tetani simultaneously recorded by spring (upper line) and by bag (lower line) to illustrate the statements made in the text, especially:—

1. The general parallelism between lateral and longitudinal indications. 2. The decline of voluntary and of direct contractions. 3. The recovery at periphery during central activity, and at centre during peripheral activity.

The time line indicates minutes. The numbers 1 to 6 point to the successive groups of contractions.

fatigue, which may indeed have subtracted from, but cannot have added to the muscular "leistungsfähigkeit." If anyone can preserve the smallest doubt in the matter, I would invite him to dismiss the doubt by experiencing a prolonged maximum endurable tetanisation interpolated between two fatigue series of maximum voluntary efforts.[1] And although direct faradisation will produce much less obvious signs of fatigue than voluntary action, it is most surprising, and I may add unpleasant, to experience the subjective local muscular symptoms which last for days and weeks after such an observation has been properly made. There is some very deep-seated difference between maximum direct contraction and maximum voluntary contraction; the former is ineffectual and injurious, the latter is powerful and harmless. I am unable to conjecture what may be the nature of the difference, but may add, in possible contribution towards its future comprehension, that the acoustic characters of the two kinds of contraction are quite distinct; the soft tone heard on auscultating muscle during voluntary contraction cannot be reproduced by faradisation at any interruption frequency that I have tried; at 20 per second a corresponding number of distinct thuds are audible; at 50 and 100 per second a musical tone of corresponding pitch is audible; but I may add further that in galvanotonus, *i.e.*, the muscular tetanus produced during the passage of a strong constant current, the muscle sound is identical with that of voluntary contraction. I am not, however, prepared to discuss the bearing of this last point upon the composite or non-composite nature of voluntary contraction.

The inference from the fatigue data above described, is an obvious one. Voluntary fatigue depends more upon central than upon peripheral change; we cannot, by forced voluntary action, unless under special conditions of experiment, cause any obvious diminution in the direct excitability of muscle, on the contrary we have just witnessed an actual increase of peripheral power during maximal voluntary expenditure; and we shall be inclined to attribute this to a double cause—to the diminishing energy of cerebral effort, and to the increasing impermeability of the motor end organ.

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[1] Mosso, by the weight method, has obtained similar results, and has explained them in a similar manner. (*Loc. cit.*, p. 123.)

The next observation, the data of which are contained in the same figure, is the converse of the preceding one. We have examined direct contraction, before and after voluntary contraction; we can now examine voluntary contraction, before and after direct contraction. If we take a few voluntary maxima before and after a period of direct tetanisation as strong as can be borne, we find no appreciable difference between the two sets of maxima.

To avoid even temporary misconception, I hasten to add that under other conditions of experiment, we can bring into evidence the peripheral expenditure provoked by central action, as well as the subtractive effect of purely peripheral action upon total or voluntary action; and that the apparent contradiction between the appearance of a *plus* effect, or of a *minus* effect, according to circumstances, is owing to the predominance of the disintegrative or of the integrative changes, which go on simultaneously, or in reverberation with each other, in the motor organ. These changes—owing possibly to the fact that depression of function at certain points, especially at the junction between nerve and muscle, can temporarily separate the central from the ultimate peripheral component—may be brought into evidence by suitable conditions of observation. We shall also find that it is equally easy to bring into evidence the disintegrative effects of direct muscular faradisation; they are manifest in every dynamographic series of directly excited contractions (*e.g.*, in fig. 4.)

The clearest evidence of the direct or peripheral disintegration produced by voluntary action, is afforded by interpolation series, *i.e.*, by taking direct tetani at regular intervals in the course of a maximal voluntary fatigue series (*e.g.*, fig. 5).

(*a*) Interpolating direct tetani at intervals (of half a minute) in a voluntary fatigue series (at 15 per minute) each effort and each interval lasting two seconds, we find that there is a decline of the direct effects parallel with the voluntary decline; this direct decrease is obviously due to a peripheral expenditure caused by the voluntary efforts, the former being at intervals so long as not to cause of themselves any appreciable fatigue.

(*b*) Repeating the experiment with submaximal voluntary efforts (20 kg.), a precisely similar result is observed; the direct tetani, interpolated at intervals of half a minute,

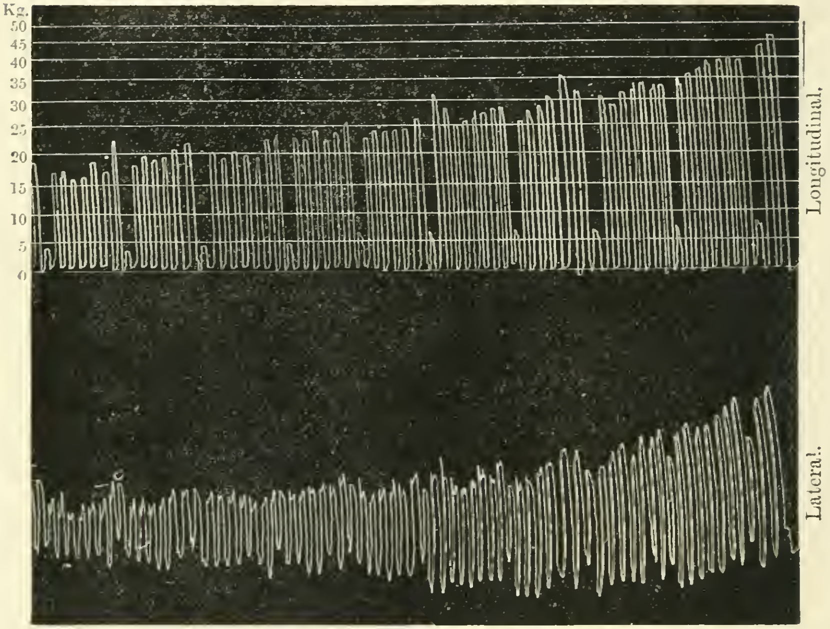

FIG. 5.

Fatigue series of maximum voluntary efforts, with interpolated maximal direct tetani. The upper line is that of the dynamographic spring, giving the longitudinal effect; the lower line is that of the bag recorder, giving the lateral effect. The interpolated direct effects exhibit in the longitudinal record a decline of direct "workability" caused by voluntary action; this decline is legible with difficulty from the lateral record.

gradually decline; the fact is demonstrative of peripheral expenditure caused by normal submaximal voluntary action.

(*c*) The converse experiment—maximal voluntary tetani interpolated at long intervals in the course of a fatigue series by direct faradisation—exhibits a comparatively small decline in the voluntary "workability" of the total cerebro-muscular organ, *i.e.*, a subtractive effect upon the whole, due to the expenditure by a part.

Thus we have seen by a few simple experiments:—

I. Falling voluntary maximum during action.	*i.e.* Evidence of total expenditure caused by total action (central and peripheral).
II. Recovery of voluntary maximum during inaction.	*i.e.* Evidence of total restoration during total rest.
III. Falling direct effects during voluntary action.	*i.e.* Evidence of peripheral expenditure caused by total action.
IV. Recovery of direct effects during voluntary action.	*i.e.* Evidence of peripheral restoration during total action.
V. Falling voluntary maximum during direct action.	*i.e.* Evidence of peripheral expenditure, causing a diminution of total action.
VI. Recovery of voluntary maximum during direct action.	*i.e.* Evidence of total restoration during peripheral expenditure.
VII. Falling direct effects during direct action.	*i.e.* Evidence of peripheral expenditure caused by peripheral action.
VIII. Recovery of direct effects during direct inaction.	*i.e.* Evidence of peripheral restoration during peripheral rest.

All these facts carefully considered almost speak for themselves, but in order to expose them more conveniently to examination and criticism, we may supplement the above summary by some comments and indications to illustrations given in this paper.

Facts (I.) and (II.) are matters of common experience, but not inclusive of any precise knowledge whether the effects are central or peripheral or both; the inferences as to the disintegrative and reintegrative causes of the effects are matters of common consent, but not inclusive of any precise knowledge whether these molecular antecedents are central or peripheral or both. These two fundamental facts have not been specifically illustrated by any figures in this paper; every dynamographic record exhibits the declining maximum by repeated effort, and the recovering maximum during rest.

Facts (III.) (V.) and (VII.) are localised phenomena of disintegration. Facts (IV.) (VI.) and (VIII.) are localised phenomena of reintegration. We shall probably find it convenient to refer these two opposite changes to a hypothetical force-producing substance, and to designate it by the term introduced by Hermann, "*inogen.*"

Of the group of facts demonstrative of disintegration of inogen, (III.) and (V.) are illustrated by the upper lines of the interpolation series in figs. 5 and 6; (VII.) by the faradic declines in the upper line of fig. 4.

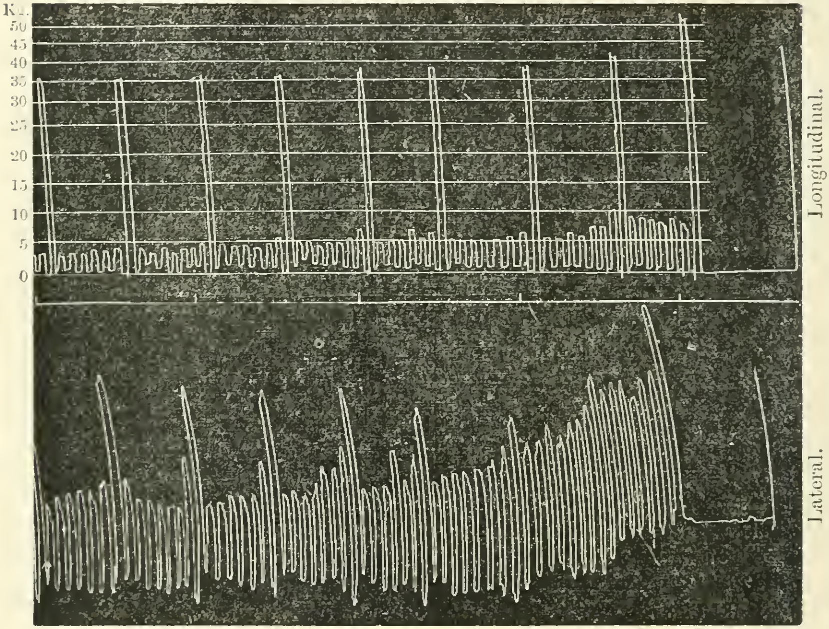

FIG. 6.

Fatigue series of maximal direct tetani, with interpolated maximum voluntary efforts. The upper line is that of the spring, the lower line that of the bag recorder. In the longitudinal or dynamic record by the spring the interpolated voluntary maxima exhibit a slight decline caused by peripheral action; in the lateral or rigor record by the bag the effects undergo a slight augmentation.

Of the group of facts demonstrative of reintegration of inogen, (IV.) and (VI.) are illustrated by the alternate groups of voluntary and faradic contractions of fig. 4, and the same figure incidentally illustrates (VIII.), as well as (I.) and (II.) *a fortiori*, *i.e.*, recovery is manifest during incomplete rest as regards (VIII.) and (II.), and (I.) is present as a matter of course.

Finally we may remark that (III.) the direct effect of voluntary exertion, considered in conjunction with (IV.) the direct recovery during voluntary exertion, afford the strongest possible proof that disintegration and reintegration of inogen can proceed simultaneously and coincidently.

Before proceeding further, we must take this opportunity of describing the instruments and methods employed in the investigation. Their consideration will apparently lead us away from the main problem into a technical side-issue, which, however, will ultimately prove to have afforded a side view of that problem, and bring us back to it with added knowledge of central *versus* peripheral effects, derived from a clearer comprehension of the significance of "contracture."

§ 7—Instruments and Methods.

The induction coil used for faradisation was a standard Du Bois-Reymond apparatus made at Berne under Professor Kronecker's supervision, and graduated in units of intensity—a grove cell in the primary circuit. The electrodes were of woven wire covered with gelatine, one "indifferent" electrode of large area was strapped to the leg, the other "exciting" electrode of smaller area was strapped to the forearm or over the median nerve; these electrodes even without the strap adhere to the skin, and cannot shift during experiment. The strength of faradisation is given in the units of Kroneckers' scale; the figures given do not, however, imply that a comparison is possible between the effects of successive observations, the exciting electrode used for nerve excitation being of smaller area than that for muscle excitation. The important point was, however, to be assured that the current strength remained constant during an observation, or to know how much it varied. A reflecting galvanometer was therefore occasionally switched into the secondary circuit, and the resistance measured in the usual way by a Wheatstone's bridge. The gelatine electrodes gave, however, a much smaller cutaneous soakage effect than the ordinary wet electrodes.

The excursions of levers were recorded on a smoked cylinder carried by the hour axis of an American clock, or by an axis connected with the hour wheel and revolving once in a quarter of an hour.

The dynamograph used consists of a short stiff spring yielding very little to the grasp of the hand, so that the length of the contracting muscles undergoes very little alteration. The deflection of the spring is recorded by a very long lever, and the value of the ordinate in kilogrammes is known from a preliminary graduation. The apparatus of Donders and Van Mansfeld is described in Hermann's Physiologie (vol. 1, p. 9). The upper extremity is placed with the arm vertical and the forearm horizontal, the internal condyle being in a cup C, the shoulder against a crutch A, the weight is suspended from a bracelet B, and to take an observation is suddenly lifted from below, the forearm springs upwards, the value of the excursion being read by an index on the scale.

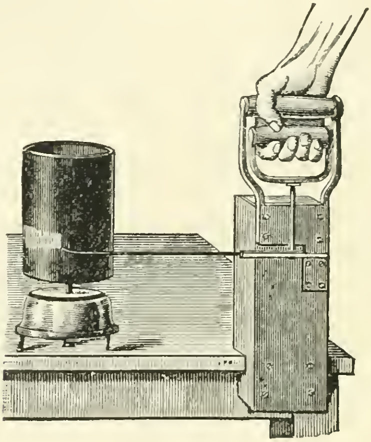

Fig. 7.

The Dynamograph.—A short stiff spring connected with a handle and slightly deflected by the grasp of the hand. The deflection of the spring is recorded by a long lever (represented here as much shorter than it is in reality).

In consequence of the great inconvenience of taking exact readings, and of reviewing columns of figures rapidly, I employed, in addition to the index (which does not belong to the instrument as originally described), a second lever in a plane at right angles to that of the index, the other end of which recorded the excursions of the arm on a smaller scale on the hour cylinder. Successive observations, the times at which they are taken, the intervals of rest and of suspension, the identity or failure of identity of the point of departure could thus be conveniently reviewed. This will be referred to as the first modification.

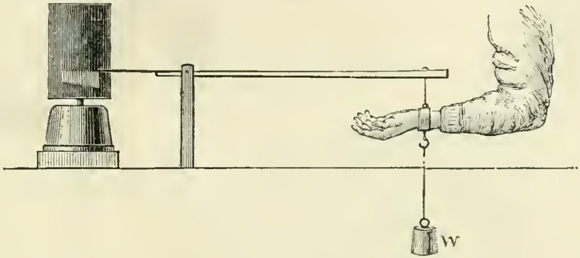

FIG. 8.

First modification of the apparatus of Donders and *v.* Mansfeldt.

A second modification was adopted in order to test the after-effect isometrically; the weight is suspended as before, and a spring and lever, the excursions of which are graduated in kilos, is placed above the wrist, a vertical rod and button serve to communicate the tension of the released forearm to the spring and lever; the button is adjusted to such a height that the head of the radius touches it when the forearm is horizontal.

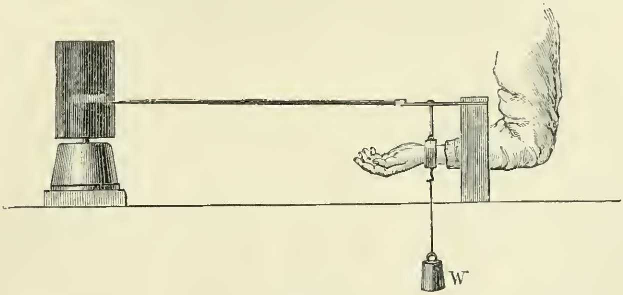

FIG. 9.

Second modification of the apparatus of Donders and *v.* Mansfeldt.

Mosso's "ergograph" and "ponograph" are described and figured in Du Bois-Reymond's Arch. 1890, p. 93, 119. The instrument here shown consists of a weight, and an arm holder, &c., to fix the hand and forearm. The weight W, is raised by the flexors of the middle finger, its rise being recorded by a pen rubbing against a vertical cylinder; used by itself the weight and pen constitute the essential parts of Mosso's ergograph. To be used as a ponograph, the weight W_1, is reduced, and joined to a horizontal lever so as to raise a second weight W_2, which on reaching a certain height drops, and the first weight springs up alone; the lever catches on again each time this first weight is let down.

The apparatus for recording the hardening of muscle consists of a thick elastic bag fixed to the forearm by a broad leather band and communicating with a tambour arranged to record its indications below those of the dynamographic spring. The value of the indications of the spring and of the bag may be shortly characterised by saying that a name to the former instrument should terminate in—meter, to the latter in—scope.

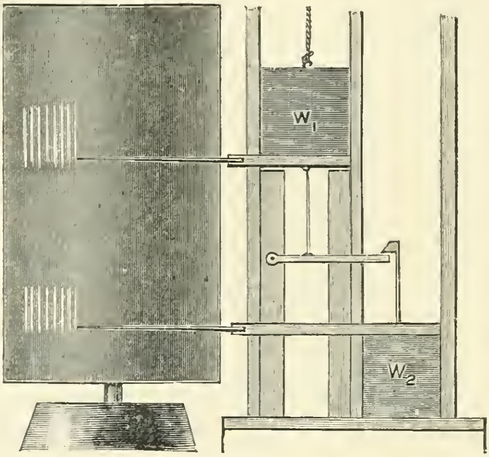

Fig. 10.

Modification of Mosse's apparatus ("Ponograph").

We may not convert, and in fact never have converted, the ordinates of the lateral indications into a series of numbers, we have at most derived from these indications rough fractional expressions to give form to the expressions "greater" and "smaller," "rapid decline" and "slow decline"—to say for instance whether one effect is about $\frac{1}{8}$, $\frac{1}{4}$, $\frac{1}{2}$, another effect; in all other comparisons between lateral indications we have been careful to observe identity of circumstances. This precaution was particularly observed in the case of peripheral faradisation series of muscle and of nerve, in which lateral records were the most convenient muscular language from which to read increased effect or diminished effect. But in forming fractional estimates of the decrements or increments, we always referred to the simultaneously recorded testimony of the indications of longitudinal tension recorded by the dynamograph. And as the spring used for voluntary maxima was too strong to afford a convenient range of deflections by peripheral faradisation, I substituted in this examination a lighter spring arranged to give an ordinate at its recording extremity about five times as great as that of the voluntary dynamograph; this lighter spring being graduated from 1 to 15 kg., was suitable for the comparison of nerve with muscle faradisation by the intercalation method.

The ordinates of the two kinds of indications were carefully compared because I soon found that the hardening and the force do not vary precisely together in fatigue effects, and therefore wished to graduate the bag sufficiently to utilise it as a rough metre. If a simultaneous record (by spring and by bag) is taken of a declining series of maximum voluntary efforts, a very interesting relation is witnessed between the two series. The spring series declines far more rapidly than the bag series. This relation signifies that with smaller external effect there is a larger internal effect, the muscle hardens more but does less, and it is natural to see in this alteration a manifestation of true peripheral fatigue. The circumference of the limb also increases in the course of prolonged observation; this may be due to increased vascularity, or to contracture, or to both (it is of course necessary to exclude an apparent increase due to warming of the air in the bag, this is done by a T piece and tap, so that air pressure can at any moment be equalised with atmospheric pressure). It is difficult to separate these two factors, but as regards each individual excursion we can be in no doubt, whatever level it starts from, it is a measure of the thickening and hardening of the flesh surrounded by the leather strap. It seemed probable that simultaneous records of muscular hardening and of external effect taken on a more rapidly travelling surface might bring the divergence into stronger evidence. For it might be expected that if in the state of fatigue there is more hardening to less result, there would also be a prolongation of the former effect. This proved to be the case.

§ 8—Distinction between Longitudinal and Lateral Effects.

Comparing the simultaneous records of bag and spring, we find that the hardening slightly precedes and outlasts the manifest effect in fresh muscle, and *that the difference in the latter case is greatly exaggerated in fatigued muscle.* Figure 11 is the reproduction of a maximum voluntary effort before and after a fatigue experiment, and upon examination will be found to exhibit just what we anticipated. The first effort was about 50 kg., the muscular hardening slightly outlasts it. The last effort was about

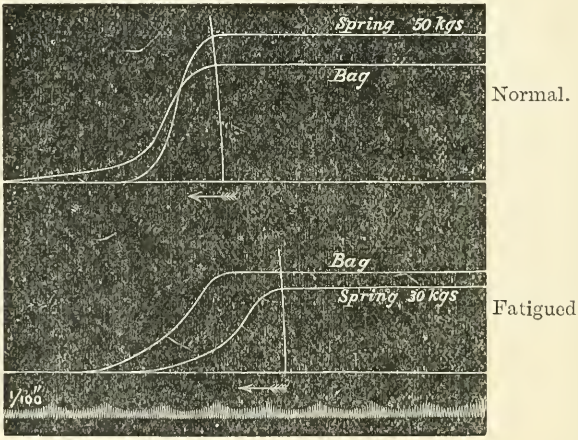

Fig. 11.

The upper two lines are the simultaneous tracing of the termination of the longitudinal or dynamic effect, and of the lateral or rigor effect produced by maximal voluntary contraction in the fresh state. The lower two lines are a similiar tracing of the effects produced by maximal voluntary contraction in the fatigued state.

The points illustrated in these two pairs of effects are:—1. That the terminal points of the effects are practically simultaneous in the fresh state, while in fatigue the lateral effect outlasts the longitudinal effect. 2. That the magnitude of the longitudinal effect is relatively more diminished than that of the lateral effect in the fatigued as compared with the normal state.

It will be seen from the next figure that the asynchronism between the terminal points of the longitudinal and lateral effects is not brought out by direct faradisation.

The time is marked in hundredths of a second. The grouping is due to "beats" accidentally produced between the time lever and the fork.

30 kg., the muscular hardening lasts much longer than the deflection of the spring; it may also be noticed that the hardening as compared with the actual deviation of the spring is more considerable in the second than in the first case; the muscle hardens less it is true, but in comparison with what it does, it hardens more.

This is *prima facie* evidence of peripheral fatigue caused by voluntary action, a "contraction remainder" such as that with which we are familiar in the laboratory, and which was first carefully studied by Tiegel and by Kronecker. If, as seemed probable, the contracture were a peripheral event occurring in the muscle itself, it should also be manifested in a prolongation of the contraction or of the tetanus provoked by direct excitation. Accordingly the record of half-a-dozen single muscular contractions (bag method; hardening of the forearm) was taken before and after a fatigue series during which the maximum effect fell 50 per cent. The contractions were higher and a little longer (see fig. 3), but the difference was not at all comparable to the effect described above.

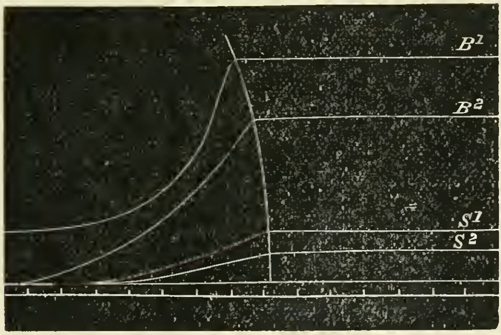

FIG. 12.

Simultaneous bag and spring tracings from the muscles of the forearm excited by direct faradisation; to shew absence of the prolongation of lateral effect with this mode of testing.

B^1 Lateral effect. S^1 Longitudinal effect (before fatigue).
B^2 Do. S^2 Do. (after fatigue).

Contrast with figs. 11 and 14, which show the prolongation of lateral effect on the same muscles, under similiar conditions, but with voluntary contraction.

The experiment was repeated with a short period of faradisation (2 sec.) in order to obtain a greater amount of hardening than was possible with a single twitch. The faradic tetanic hardening, so far from being less, was a little greater in the fatigued than in the normal state, but its period of maintenance of contraction was very slightly increased.

The peripheral explanation, which seemed so probable, is thus disproved, and we are driven over to the more improbable supposition that the maintained hardening, must be in large part, if not wholly, central; improbable though the supposition appears at first sight, we must imagine that the fatigued centre maintains an effect at the periphery, which manifests itself in a period of maintained muscular hardening without *corresponding* maintenance of dynamic effect. The word "corresponding" has been underlined because, as we shall see by other experiments, there is in voluntary fatigue a certain degree of the latter, which we shall see cause for attributing to sluggishness and less prompt cessation of central action. Another point to be recognised at present is that the two objective phenomena—hardening and dynamic result—are not necessarily associated in a parallel manner in voluntary and in faradic tetanus; the hardening is much greater in proportion to the dynamic effect in the latter than in the former; *i.e.*, in faradic tetanus we have nothing like the dynamic effect which is produced in a voluntary tetanus which produces the same or even a smaller amount of hardening, and a similar relation is brought out in the course of voluntary fatigue, the dynamic effect falls more rapidly than the rigor effect; the latter may in fact be absolutely increased during fatigue, as is illustrated by fig. 3.

The maintained rigor effect which is so striking a feature in voluntary fatigue, is an exaggerated sign of this dissociation between the lateral and the longitudinal effects. I do not, however, say that the prolonged maintenance of the lateral or rigor effect is *entirely* of central source, but that it is mainly central. A similar but much smaller effect can be demonstrated as a purely peripheral phenomenon, but even in this detail the principle that normal fatigue phenomena are in major part central, in minor part peripheral, seems to assert itself.

An objection may be raised—we must remember that in the comparison just made, the two terms have been maximal voluntary and maximal faradic, and that the difference may be due to or exaggerated by the normal disparity between these two maxima as regards the longitudinal effect. It would be desirable to establish a further comparison between maximal faradic and submaximal voluntary tetani of equal dynamic values, and thus, see whether the prolongation found with maximum voluntary tetanus also holds in the case of submaximal action. As a matter of fact, it does so.

The peculiar after-effect developing in the course of fatigue, and manifesting itself as a hardening without corresponding shortening, deserves closer study; it is a loophole through which we may obtain a glimpse of the intimate nature of voluntary contraction and of contracture. It may sometimes be noticed that before the relaxation actually occurs there is an *augmentation* of hardening which is without effect upon the dynamogram; this terminal incident becomes more and more pronounced with increasing fatigue.

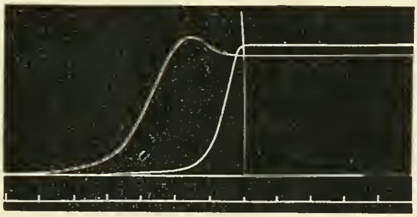

FIG. 13.

Terminal augmentation of the lateral effect associated with its prolongation in consequence of fatigue. The tracing is otherwise similiar to those given in fig. 11, and taken in a similiar manner.

How can we account for the phenomenon? We may first imagine that the series of changes in the conversion of potential to dynamic energy in the muscular contraction which, in the normal state, follow each other so rapidly as to be separately undistinguishable, are scattered and separated when the process becomes sluggish. When the fibre contracts it becomes broader and shorter, and raises a weight; if it be prevented from shortening by a strong spring, it becomes harder and more tense, and deflects the spring. When the fibre relaxes it becomes narrower and longer, and lets down the weight; or it becomes softer and less tense, and ceases to deflect the spring. The internal muscular change is a change of potential energy or tension, the external effect manifested by the weight or by the spring, is the dynamic effect.[1] In the contraction the lateral alteration (broadening or hardening) *precedes* the longitudinal effect (shortening or increased tension). In the relaxation the lateral alteration *outlasts* the longitudinal effect. In fatigue it does so more than in the normal state. The phenomenon immediately under the influence of nerve is the lateral effect, which again is the immediate antecedent of the longitudinal effect.

"*Contracture*" is a persistence of the lateral effect which, in much less degree, entails persistence of the longitudinal effect. In "Rigor" there is considerable lateral effect (hardening) but scarcely any longitudinal effect. In direct faradic tetanus there is comparatively great lateral effect to small longitudinal effect.

With voluntary contraction there is comparatively smaller lateral effect to greater longitudinal effect; and we find that the ratio between *lateral* and *longitudinal* effects is expressible as a greater fraction in the fatigued than in the normal state. These points are exemplified in fig. 11, and to a still more marked degree in the following numbers, which express the fraction $\frac{\textit{longitudinal}}{\textit{lateral}}$ in various cases[2]:—

Maximal voluntary contraction	40 kg. : 25 mm.
,, faradic ,,	8 ,, : 25 ,,

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[1] Properly speaking, in muscular contraction the sequence is,
1° Chemical potential energy.
2° Mechanical potential energy (Spannkraft, vis tensionsis).
3° Mechanical kinetic energy (vis viva measured by work).
The term "dynamic" is, however, equally applicable to (2) and to (3); we measure (2) by the spring isometrically; we measure (3) by the weight isotonically.

[2] Ratios in each pair are comparable; ratios in different pairs are not strictly comparable, although they do exhibit rough average relations.

Maximal voluntary contraction (fresh) 50kg. : 32 mm.
,, ,, ,, (fatigued) 30 ,, : 26 ,,
Submaximal voluntary contraction (fresh) 20 ,, : 13 ,,
,, ,, (fatigued) 20 ,, : 16 ,,
Submaximal faradic contraction (fresh) 10 ,, : 15 ,,
,, ,, (fatigued) 5 ,, : 15 ,,

We may provisionally, *i.e.*, for the purpose of analysis and experimental insulation, imagine as distinct the lateral and longitudinal effects. But the question is very complicated, and we are in danger of losing the main phenomenon from view. I therefore repeat and emphasize the fact that there is a great contrast between the voluntary muscular hardening in the fresh state, and the voluntary muscular hardening in the fatigued state, the latter has a period of maintenance, or even of increase, which residual effect is not brought into evidence by direct excitation. I see no escape at present, therefore, from the inference that the residual effect must be a residual discharge from the fatigued centre. And let no one imagine that it is by any possibility an experimental error. It is a coarse and obvious difference, lasting for one-tenth second, between the descent of the spring and the relaxation of the muscle. Perhaps it is a contraction of antagonists, but how is it that nothing of the kind is to be seen in the fresh state? It is entirely a fatigue phenomenon—a residual molecular effect at the periphery, due to a residual effect at the centre—an increase of lateral tension, such as we have in rigor or contracture, without *corresponding* increase of longitudinal tension. But again, it is a most surprising fact that this rigor effect at the periphery should not be elicited by peripheral excitation, but only by central excitation.

As far as can be seen at present, it is a phenomenon more closely related to the "contracture" of clinical medicine, which is a peripheral phenomenon caused from the centre, than to the "contracture" of the laboratory, which is a purely muscular phenomenon. But the common component in both forms of contracture is the small proportion of the total voluntary contracture or central contracture, which is really local, and can be brought into evidence by direct excitation. It is said that clinical contracture disappears at death unless it has been of very long standing, and that it is often resolved during sleep and during anæsthesia, admitting this, we clearly recognise another illustration of the major central and minor peripheral components in the total effect.

But this does not exhaust the experimental conditions under which this point can be placed and examined. We may investigate the effect of peripheral fatigue upon the maximal voluntary effort, and discover whether the rigor effect, which, as we have seen, is mainly central, is or is not manifested when normal central discharge supervenes upon a fatigued muscle. And we must see whether the effect is not manifested by peripheral excitation of nerve as well as by direct excitation of muscle ; it is, in fact, conceivable that it may be in part a motor end plate effect not manifested when the muscle and intramuscular nerve are excited, but manifested when only nerve is excited.

I have not yet found time to investigate these two points ; we have, however, evidence enough already to warrant the conclusion that an increased duration of the lateral effect is a sign of central fatigue, while an increased magnitude of that effect, as compared with the magnitude of the simultaneous longitudinal effect, is a sign of peripheral fatigue. I hope to return to this matter and to study it in closer detail ; for the present, I will only indicate as one of the chief facts under this head, that the second named feature, and not the first, is manifested by excised frog's muscle, directly excited. A frog's gastrocnemius is arranged so as to yield a simultaneous record during fatigue, (*a*) of the isometric longitudinal effect, (*b*) of the lateral effect. For the record *a* the muscle was made to pull upon a strong spring; for record *b* it was made to raise a light spring, by which it was compressed, and both springs were made to record their deflections on the slow cylinder. The result is illustrated in fig. 14.

Thus we have a disproportionately large lateral effect as an undeniable peripheral phenomenon, and we may admit that in all probability the disproportion developed in voluntary fatigue is a sign of peripheral modification distinct from the prolongation of effect, which is a sign of central modification. If the term "contracture" is to be made use of, the first event must be characterised as peripheral contracture, the second as central contracture. We have already seen that in voluntary contraction the proportion $\frac{\textit{longitudinal effect}}{\textit{lateral effect}}$ is greatest, and that in direct contraction it is least. We have also seen above that the proportion falls rapidly in purely peripheral fatigue, but less rapidly in mixed voluntary fatigue. These several facts, collectively considered, appear to me to afford some insight into the nature of the complex phenomenon of "contracture," and of the varying relations between contracture and contraction by normal and by artificial muscular stimulation. The essential point appears to be to distinguish between the lateral and longitudinal effects, and to study the relations between them by simultaneous records taken under varying circumstances. As far as I know, this has not previously been done.

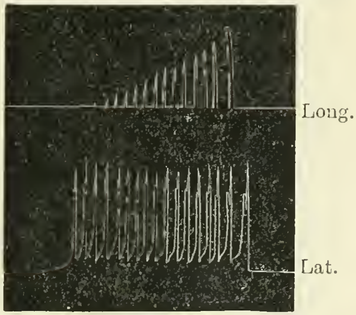

FIG. 14.

Simultaneous tracing of longitudinal and lateral effects taken from a frog's gastrocnemius on a slow cylinder. The longitudinal effects exhibit a far more rapid decline than the lateral effects.

It was obviously necessary to examine whether there is also any maintenance of the dynamic effect; we have indeed seen that there is no such maintenance at all corresponding with the maintenance of lateral effect, but the question now to be put is whether there is any maintenance at all of the dynamic effect, and if any, whether it is of central or of purely peripheral mechanism. We shall find when we come to the consideration of effort effects (§ 10) that this point must be taken into account in interpreting the results. The experimental analysis of the ending dynamic effect is, however, much more delicate than that of the ending rigor effect, and for the sake of brevity is best presented by the following comparisons.

(*a*) Comparing the returns to the zero line of a dynamographic spring from its position of maximum deflection by

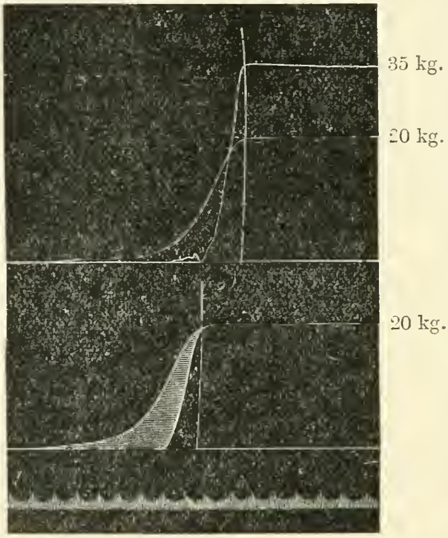

FIG. 15.

Tracings to illustrate the prolongation of the longitudinal or dynamic effect caused by fatigue.

The upper pair of lines give the returns from voluntary effects on a strong spring. 1. In the normal fresh state (35 kg. effort). 2. In the fatigued state (20 kg. effort). The lower pair of lines give the returns from a submaximal voluntary effort of 20 kg. in the fresh state and from a maximal voluntary effort of 20 kg. in the fatigued state; the area between the steep return in the fresh state and the more gradual return in the fatigued state has been shaded in order to shew the difference in the two cases.

voluntary contraction in the normal state, and in the state of fatigue caused by voluntary action, it is found that the return is more rapid in the first than in the second case.

But the deflection is smaller than in the first; it was therefore necessary to determine further—and this has been done—that the decline in the second case is also less rapid than that from an *equal* deviation caused by submaximal contraction, *i.e.*—(*b*.) Comparing the return from a maximal contraction in fatigue with that of submaximal contraction of equal dynamic value in the normal state, it is found that the former is more gradual than the latter.

It was also desirable to see whether the terminal asynchronism of lateral and longitudinal effects is manifested by the weight method combined with the bag-recorder. The examination of this point led to some interesting results; the dynamic effect in this case begins to drop after the lateral effect has begun to decline. I will not follow the considerations into which this fact leads, as the matter soon passes beyond the bounds of a neurological item; it need only be remarked that once recognised, the phenomenon is very obviously reasonable; the muscular tension is more than sufficient to hold up the weight (2 kg.) which therefore does not begin to drop until the tension has fallen below that adequate amount. A similar asynchronism exists also at the outset of a contraction, the tension begins sooner than the weight rises, the latter event does not begin until the tension has reached the adequate amount.

As to the point which we are examining on account of its neurological significance, the event is as was anticipated, the rigor effect and in less degree, the dynamic effect decline more slowly in fatigue than in the normal state. I have not yet made methodical experiments as with the dynamograph to determine in what degree the effect is central or peripheral or both. I think, however, that the proof already given by the spring method may also be held to cover the effects obtained by the weight method. It will, however, be obvious that the relation between lateral and longitudinal effects demands close and minute analysis; up till now I have only been able to examine the matter in so far as it afforded differential indications of central and peripheral phenomena.

§ 9—The End-plate Effect.

In the report from which quotation has been made above, it was urged as a general conclusion that the junction between nerve and muscle is a weak link in the neuro-muscular element, and the first point to show signs of failure in fatigue, in nerve-degeneration, at death, and in intoxication by drugs of the curare type. The conclusion was based mainly upon frog muscle observations, but in part upon human muscle observations.

On returning to the examination of this point I find that it is possible to demonstrate signs of an end-plate effect in nerve faradisation and in normal voluntary action. Nevertheless it is necessary to admit that the evidence is not conclusive, and that it must be considerably discounted owing to the complex conditions of experiment.

The best obtainable evidence is that supplied by intercalation series; direct muscular faradisation is applied at intervals in the course of a fatigue series of nerve faradisation effects, or *vice versâ;* nerve faradisation effects are intercalated in series of direct effects; the former being obviously the series from which positive information is to be expected. But the control experiment is required because the intercalated muscular effects may decline less rapidly than the nerve series in consequence of fresh muscle being each time called into action by the direct excitation. If, however, we find *under precisely similar conditions of experiment* that the intercalated nerve effect declines as fast as or faster than the muscle effect, we have good reason to attribute the less rapid fall in the previous case to an end-plate effect. To demonstrate the existence of the effect in voluntary action a similar mode of observation is followed, pairs of nerve effects and of muscle effects being intercalated at intervals in a voluntary fatigue series.

In taking intercalation series of nerve and muscle it is not possible to use the coil at the same strength in both cases, nor is it possible to shift the secondary coil from one position to another with sufficient rapidity and accuracy. It is necessary to equalise matters by using an additional resistance of 10 to 50,000 ohms in the nerve circuit.

A very obvious effect at the outset of an experiment marks the fatigue decline, the effects increase (*a*) by diminishing skin resistance (*b*) by increasing excitability.

In establishing a comparison between the effects of the two modes of peripheral excitation it must be recognised that the two phenomena are impossible to insulate to the same degree on human nerve and muscle as on excised nerve and muscle. (1) We cannot accurately compare the excitability of nerve with that of muscle, different current strengths are used in the two cases, and even if we use the same current strength, the distribution of current is necessarily dissimilar. (2) We do not call into play identical portions of muscle when, *e.g.*, we apply excitation to the median nerve and to the muscles of the forearm. All we may accomplish in the comparision is to bring into action a certain amount of the same muscle in the two different cases. (3) Even in that common portion we have a comparison between effects on nerve *and* muscle in the one case versus nerve in the other. There is then no reason for surprise if an end plate effect is not so clearly demonstrable on intact human nerve and muscle as on the isolated tissues of animals; on the contrary, the matter of surprise will be if any such effect can be brought into evidence in spite of the three veiling circumstances just enumerated.

All things considered, I am not inclined to lay much stress upon any data which I have obtained from human muscle. Although these data do, as far as they go, support the idea that prolonged activity can cause a block at the junction between nerve and muscle, I do not—in view of the complex experimental conditions adverted to above—think that they are clear enough to be utilised by themselves as a premise in the argument. This reservation does not, however, apply to the data obtainable on isolated frog's muscle; these are clear and definite, and sufficient to justify the use of the "end-plate effect" as part of an hypothesis.

§ 10—Measurement of Effort.

Most persons have at one time or another had some such experience as this—he goes to lift an empty can supposed to be full of water and jerks it high into the air; in consequence of the exaggerated anticipation of weight an excessive nervous effort has been emitted. Some kind of measure of the nervous emission is obtained in the experiment of Donders and Van Mansfeld, in which a weight suspended from the wrist of the horizontal forearm, is suddenly removed; the arm springs up by the excess of muscular contraction, and the spring can be measured by suitable apparatus under various conditions. A record of the same phenomenon can be obtained by means of an ingenious instrument devised by Mosso and termed by him the ponograph or effort-measurer. In principle this is a recording slider to which a weight is attached, the weight is raised and caused to drop early in the muscular contraction, the excursion of the liberated slider gives measure of the magnitude of effort made to raise the weight.

I have made a considerable number of observations of this kind with the apparatus of Donders and Van Mansfeld, and more recently with apparatus similar in principle to that of Mosso, and using the same muscle and the middle digit which lend themselves best to localised faradisation. The apparatus shown in fig. 8 is contrived on the same principle as Mosso's ponograph, to allow a weight to be raised and to be suddenly reduced early in the contraction, the upward throw of the liberated portion being utilised as the indication of the energy of contraction put forth to raise the whole weight.

Experimenting with the apparatus of Donders (which was originally devised for an investigation of the elasticity of human muscle) the fact which most concerns us at present is that the upward spring of a fatigued arm is greater than that of a fresh arm. Experimenting with the ponograph it has been found by Mosso that a succession of voluntary elevations of the suddenly released slider, progressively increase.

Mosso infers from this increase that a greater nerve-impulse is sent to a fatigued than to a fresh muscle; if this be so the effect is a manifestation of peripheral fatigue. But the facts do not necessarily confine us to this conclusion, and we may equally well attribute the effect to the greater sluggishness and consequent less sudden cessation of the voluntary emission; if this be so, the effect is a manifestation of cerebral fatigue. Further evidence is however required before we can choose between these two opposite conclusions; the facts as they stand may prove to belong to either. The observation already described of the maintenance of a rigor effect, and that about to be described of a smaller but obvious dynamic effect at the periphery in consequence of residual central action, furnishes such evidence, and goes far to establish the view that the increasing release effect is indicative of central prolongation of discharge. We have seen that there is a striking prolongation of the lateral effect in voluntary fatigue, and that the prolongation is owing to a prolongation of central discharge. We have also seen, but far less distinctly, that there is a prolongation of the longitudinal or dynamic effect at the periphery by the same cause. This point was investigated as follows, and it will be seen with a positive result although for the sake of completeness must also be described in what particulars the result was negative, viz., especially the failure to obtain any evidence that the discharge *begins* to cease any sooner in a fatigued than in a fresh centre.

§ 11—Active "Let-go" Experiments.

I did not at first see any way of experimentally testing this point; how indeed was it possible to signal the moment of cessation of a voluntary discharge, so as to get at data indicating whether the period of maintenance varied with fatigue? The point can, however, be experimentally investigated in a very simple, although not very searching, manner, by means of a double dynamograph simultaneously acted upon by the two hands.

First, it was necessary to determine whether the grasps of the two hands simultaneously willed and inhibited, are actually executed together, or whether there is any irregularity or asynchronism, between the records of the two simultaneously willed movements. Secondly, it was advisable to determine whether voluntary fatigue of one arm (*i.e.*, of the cerebro-muscular apparatus of one side), causes any voluntary fatigue of the opposite side.

Briefly stated, the examination of these two preliminary points showed (1), that the positive and negative acts simultaneously performed on the two sides are simultaneous with an average error not exceeding ± ·02″. (2), That fatigue of one arm does not cause any demonstrable fatigue of the opposite arm.

These two points having been determined, we are in position to make the comparison. One arm is fatigued; a synchronous volitional effort is made on both sides. The indications of the fresh side were used as the term of comparison to which the indications of the fatigued side are referred, in the expectation that the fatigued side might be found to let go later than the normal side. Observations were taken in sufficient number to eliminate experimental errors, and a comparison of the points of relaxation of both hands, showed that the average asynchronism in the normal condition and with fatigue on one side, was ± ·02″, without any ascertainable constant difference attributable to fatigue. But closer examination of the record of the liberated spring showed that the "diastole" was more rapidly accomplished by the normal than by the fatigued hand, the spring was let go more slowly and gradually in the latter than in the former case. It goes without saying that the deviation from which the spring returned to its position of rest was smaller on the fatigued than on the normal side, and that the return was consequently compared with return from an equal deviation made by the normal hand—as has already been described above (see fig. 14).

§ 12—Passive release Experiments.

The conditions of observation with the Donders' and Van Mansfeld's apparatus are as follows:—During the sustention of a weight by the horizontal forearm, the length of the biceps is the resultant of two equal and opposite factors, (*a*) the extending force of the weight (including that of the forearm), (*b*) the elastic force of the extended muscle. On suddenly diminishing factor (*a*), the arm springs up to a new position of rest by excess of factor (*b*). It has been shown by D. and V. M. that this upward spring is greater in the fatigued than in the normal state, and we desire to learn whether this increased effect depends upon a greater motor energy emission to fatigued muscle, or upon a prolongation or less rapid cessation in the emission of energy. This was the specific question on account of which the experiments were undertaken, and the modification about to be described, and sketched above in Fig. 9, was devised with that special end in view.

The arm and forearm are placed against a support adjusted so that the arm is vertical and the forearm horizontal; the head of the radius just touches a button and short vertical rod connected with a stiff horizontal spring, the small deflection of which is magnified and recorded by a light lever; a weight suspended from the wrist is suddenly released as already described. The deflections of the spring indicate the dynamic effect, and are not found to increase with fatigue. This is at first sight a paradox, in comparison with the increasing excursion of the released forearm. We have the same dynamic effect on the spring (*i.e.*, under approximately isometric conditions), but a larger excursion of the limb (*i.e.*, under conditions approximating to isotonic) in the fatigued as compared with the normal state; and the most natural supposition to account for the paradox would seem to be that the dynamic effect declines more gradually on removal of the peripheral counterpoise in the fatigued than in the normal state. But I have failed to obtain any experimental verification of this supposition under the conditions described, and have not found the isometric release deflection to be accomplished appreciably faster in the normal than in the fatigued state.

If the isometric release effect be taken with different weights, *e.g.*, 3 kg., 5 kg., the deflection of the spring will be found to correspond almost precisely with the deflections produced by the applications of weights, 3 and 5 kg. respectively, whether the limb is fatigued or not. This is as might be expected, the elasticity of the brachial flexors is necessarily equal to the extending weight, and the paradox is now seen to be susceptible of another explanation.

In the state of fatigue a greater and more prolonged lateral effect is associated with a given longitudinal effect than in the fresh state. The swelling of the forearm associated with a longitudinal contraction or elasticity of, say 5 kg., is greater and more prolonged in the fatigued than in the normal state, and when the weight is suddenly removed or reduced the excursion of the liberated lever is also greater.

It is quite intelligible that either a greater or a more prolonged lateral effect, associated with a constant longitudinal effect, should give a greater excursion of a suddenly unloaded lever, but an equal deflection of a dynamometric spring.

With reference to the immediate point at issue—whether the increasing release excursions of the forearm in Donders' and Van Mansfeld's experiment, or of the finger and slider in Mosso's ponograph observations, are the signs of central or of peripheral changes, the answer must remain much less certain than could be desired. The *prolonged* lateral effect is a sign of central fatigue, the *increased* lateral effect (relatively to a constant longitudinal effect), is a sign of peripheral fatigue. The increased release effect may be due to either of these two causes, but is more probably due to both conjointly. The probable conclusion is that the greater excursion of the forearm is due to a double cause, (1) to a central prolongation of lateral effect, (2) to a peripheral augmentation of lateral effect.

§ 13—Peripheral Inhibition?

A question naturally suggests itself as to the effect of the direct muscular excitation superposed upon an already existing maximum voluntary effect, or of a voluntary effort superposed upon already existing direct faradic tetanus. With regard to the first part of the question, an erroneous opinion has existed and may still exist to the effect that we can by direct faradisation *increase* the effect of a maximum voluntary tetanus, this may conceivably be the case in cerebral paralysis or paresis, but it certainly is not so in the normal subject; a maximum voluntary contraction so far from being increased is, as was first shown by Fick and has been confirmed by Mosso, diminished by muscular faradisation. This opens out the further question whether the interference is central or peripheral, and it seems to me that the lost time of the effect (0·1 sec.) is very strong evidence of reflex inhibition due to central interference, as was stated by Fick, and very difficult to reconcile with a true peripheral inhibition belonging to a class of effects represented by the well-known cardiac effects of the vagus, as has recently been urged by Mosso. The second portion of the question heading this paragraph may very shortly be disposed of; the greatest endurable faradic tetanus is much less than voluntary tetanus, and can be increased by the latter.

On investigating this point I was however surprised to find that I could not uniformly confirm Fick's datum, but sometimes obtained time-values too short to be admissible as those of a reflex action; nevertheless I am unable to find any serious ground for admitting with Mosso that the phenomenon is one of true peripheral inhibition. The observation, as originally made by Fick, is easily repeated; electrical stimulation is applied to the arm, relaxed or more or less strongly voluntarily contracted; a simultaneous record is taken of the effects as shown by the spring and by the bag; the strength of voluntary contraction upon which the electrical effects are superposed is of course recorded by the same means and can afterwards be read off on the dynamogram. As pointed out by Fick it will be obvious that the dynamic effects of electrical stimuli of constant strength will be smaller and smaller as the strength of voluntary contraction upon which they are superposed is made greater and greater, and that with maximum voluntary contraction, the electrical stimulation, if effective at all, subtracts from the effect; the plateau of the voluntary tetanus as recorded by the spring exhibits a step down instead of a step up in consequence of the electrical excitation.

Mosso measures the lost time to be $\frac{20}{100}$ second, and yet admits the phenomenon to be one of peripheral inhibition. The ergograph was the instrument used, and the induction currents were let into the muscle by a contact made by the slider at a definite point of its excursion; this was found not to inhibit the maximum voluntary lift. But it is to be remembered that the maximum lift was not the true maximum voluntary effect, and that it was quite possible for the muscle to go on lifting the weight as high as before, in spite of the direct excitation of itself or of its antagonists. To get signs of "inhibition" he used stronger currents permanently applied, and obtained permanent flexion or contracture with deficient voluntary contractions; he gives as the minimum lost time of the effect, $\frac{20}{100}$ seconds, but from the tracing on the same page (No. 42), it may be gathered that the maximum lost time may be at least six seconds. This is very suggestive of a "giving out" of the voluntary resolution of the observee, and of a very large margin allowed to the subjective factor, and although it may be freely admitted that the tracings bear an obvious superficial resemblance to stand-still of the heart by vagus excitation, stronger evidence must be forthcoming before the deeper similarity can be admitted; the vagus stand-still is a diastolic arrest, the faradic stand-still is a tetanic arrest. Mosso promises, however, to supply us with further grounds of comparison between the interference effects of the cardiac vagus and those of ordinary "motor" nerves (median), and until then we cannot properly estimate the probabilities of his contention.

At present it appears to me premature to attribute the step down to an inhibitory process in the muscle, and that the resemblance between Mosso's ergograms and ordinary vagus cardiograms is no more than a superficial one.

That is a much less recondite phenomenon, and probably due sometimes to the excitation of antagonist muscles, and sometimes to ordinary sensory inhibition, is indicated by two facts.

(1) The simultaneous record of muscular hardening shows that with the diminution of effect on the spring there is greater hardening of the forearm.

(2) Time-records of the effect show a lost time of between two and three hundredths of a second, which, as I

have urged elsewhere, is the apparent lost time of a direct effect on human muscle with the instruments ordinarily employed.

The conclusion is irresistible, that in such case we have to do with a simple subtractive effect upon the spring due to the diffusion of electrical excitation to antagonist muscles, which on entering into action cause the increased lateral tension as indicated by the bag-recorder.

But the time-data were by no means invariable, they frequently came out as described by Fick about $\frac{1}{10}$ second, and the effects were then recognisable as ordinary reflex effects ; more commonly still they came out about $\frac{1}{20}$ second, a doubtful and perplexing figure, too long for direct action and too short for reflex action ; the only explanation to be offered is the probable, but by no means proved, direct response of antagonists to the direct extensile stimulus caused by the direct action of the muscle under the electrode. This point requires, however, further and more careful investigation.

I cannot at present safely speak more definitely, and prefer to indicate as distinctly as possible what appear to me to be the uncertainties of observation, and therefore of inference in these particular experiments, which are of considerable interest in their bearing upon the question of direct muscular inhibition. Repeating Fick's observation on the abductor indicis, I cannot admit that there is no diffusion to the next intercarpal space with the strength of current necessary to produce the effect ; I also find that a reflex start, which during maximum voluntary abduction of the index finger takes the form of diminished abduction, does frequently occur, but that this is not a negative reflex *sui generis*, according to the meaning of Fick ; it is not distinguishable from an ordinary sensory reflex, which may indeed be reflex *minus* abductor action, but may equally well be reflex *plus* adductor action. And in point of fact I find, unlike Fick, that the step down with its latency of $\frac{1}{10}$ second can be produced by sensory stimulation of any part of the body, though with greater certainty and neatness by stimulation of the limb itself.

§ 14—The main position reviewed.

To return to the main question, viz., the central *versus* the peripheral share in the total motor expenditure consequent upon and therefore accompanying voluntary muscular action, let us pause to take stock of the position of affairs. Under the conditions of partnership between the central governing cell and the subordinate contractile fibre, it is not possible to exhaust the resources ("leistungsfähigkeit") of the total organ by functional demolition of either of the two elements; if the superior element be put into excessive action, it elicits the maximum of action in the inferior element, but being itself fatiguable, and separated from the subordinate element by a fatiguable intermediary, it cannot overdrive and exhaust its executive servant. That was my meaning in the expressions already quoted—"central fatigue is protective against peripheral fatigue"—"the junction between nerve and muscle is a weak link in the chain." As regards the subordinate element—the muscle, it cannot be exhausted by direct excitation, which never elicits the full measure of force that can be called out by the voluntary mandate, and however much it may have been worked directly on its own "responsibility," it remains indefinitely capable of work if the cerebral partner wills that it shall work.

A priori the most desirable relation between the two partners would be that the the maximum workability of both acting conjointly should decline in the same ratio in each. This is precisely what may be gathered *a posteriori* from intercalation dynamograms (upper lines of figs. 5 and 6).

In the first case, *i.e.*, of total conjoint action, the ratio between direct capacity and direct *plus* central capacity is seen to remain practically constant, *i.e.*, the ordinates of the voluntary fall and of the direct fall maintain approximately the relation $\frac{1}{5}$ throughout a general fall amounting to about 60 per 100 of each.

In the second case, *i.e.*, of partial peripheral action, the direct fall does not entail a proportionate diminution of the total voluntary yield; the former falls about 60 per 100, the latter at most 30 per 100.[1]

§ 15—Muscular Sense Experiments.

It was with great reluctance that I undertook experiments on the so-called muscular sense, because although to some extent admissible as objective evidence, they include a most variable and fluctuating subjective factor, and cannot without prolonged care be obtained "clean" from accidental distortion; they moreover require analysis into separate sensory components, and when that difficult analysis is performed with more or less accuracy, there still remains the subjective factor in still stronger proportion liable to modify the separate components. But the verbal importance attaching to certain so-called "data" from this source is so great that they cannot be ignored. I allude in particular to the data attributed to Bernhardt, to Ferrier and to Brunton, and I expressly omit all consideration of the more refined analysis of the modern German psychophysical school (Goldscheider, Münsterberg) as beside my present object.

By muscular sense is denoted a sum of sensations derived from the varying tension and pressure of all the motor organs and of the skin caused by movements. In any complete analysis of the phenomena the first problem is to separate the cutaneous component; the next is to separate the residue into its muscular, tendinous, ligamentous and articular components. In my first plan of investigation I thought that this analysis would be an indispensable preliminary, but I soon found that it would be possible to take muscular sense phenomena "in the lump" as is usually done, and not item by item as seemed at first to be required.

The starting points of my observations have been (1st) the statement quoted by Dr. Bastian as conclusive, that it has been clearly shown that estimations of weight can be distinctly made when muscles are called into activity by faradisation (Bernhardt) and by galvanism (Brunton and Ferrier) in the absence of all cerebral activity; (2nd) the desire to ascertain in what degree the subjective estimation of differences of weight is altered in fatigue, whether central or peripheral. It is only under the first of these headings that I feel justified in speaking at present. The observations under the second heading are, comparatively speaking, more delicate, therefore not yet numerous enough, and requiring to be supplemented, if possible, by closer analysis, especially by exclusion of the cutaneous factor. I may, however, say that as far as my results have gone, they have been in agreement with those of Fechner, who found that the discrimination fraction is unaltered by fatigue.[1]

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[1] These numbers are taken from the original tracings, of which the figs. here given are portions.

The facts under the first head are coarse and striking differences observable *in spite* of the co-existence of the cutaneous and other factors; the latter indeed are unfavourable conditions of the conclusions drawn and cannot therefore be made responsible for them.

I adopted the first of the three systems of observation described by Fechner, *i.e.*, that of "*just perceptible difference*"; neither the method of "*right and wrong cases*" nor that of "*mean error*" was expeditious enough for the purpose in view—nor necessary. Of course in the adoption of this method I sought for no very exact determination of the minimum perceptible; obviously if a value is taken with which no mistakes of discrimination are made, that value must be above the minimum, while if a minimum is taken, mistakes must be made and the method becomes that of right and wrong cases.

It appears to me that this point has not always been sufficiently appreciated, and that in addition to unavoidable sources of error, great uncertainty attaches to determinations of "just perceptible difference," owing to the fact that an unknown percentage of erroneous estimates have necessarily occurred even with differences greatly in excess of the just perceptible. Referring for instance to Fechner's "Fundamental Table," and to the differential determinations given by him with the initial weight=1,000 grammes, we find that the probabilities are about :—

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[1] Fechner's Psychophysic, 2nd edit., p. 300, "Parallelgesetz."

3 correct answers in 4 trials with the increment=50 grms. *i.e.* $\frac{1}{20}$
4 ,, ,, 5 ,, ,, =100 ,, $\frac{1}{10}$
9 ,, ,, 10 ,, ,, =150 ,, $\frac{3}{20}$
199 ,, ,, 200 ,, ,, =300 ,, $\frac{3}{10}$

and it is obvious that different observers may form impressions of discriminative "certainty" from very different proportions of correct answers, when a few trials are made, especially if the weights are lifted unequally or at unequal intervals. In my own case, and before I had sufficiently appreciated the liability to error even with differences of weight much above the "minimum perceptible," I was surprised to find how frequently I gave a wrong answer between 1,000 and 1,100 grammes, and did not consider the estimate made "with certainty" unless the weights compared were 1,000 and 1,150 grammes. I thought at first that this indicated a deficiency of sensibility, but found that other people (to their own surprise), made mistakes of similar magnitude, and was thereby led to recognise that the error was to be expected in accordance with Fechner's table of probabilities.

This point has been insisted upon because it teaches us (1) that a difference of weight to be perceived with certainty must greatly exceed what would be considered an orthodox "minimum perceptible difference"; (2) that an "orthodox" minimum perceptible difference must be determined by very numerous and regular observations—preferably by Fechner's second method (right and wrong cases), in which they are required in thousands; (3) that no reliance can be placed upon comparisons between two "certainly" perceptible differences derived from a few observations unless the contrast is very great; (4) that no reliance can be placed on "orthodox" minimum perceived differences based on a few observations. It will then be understood that I am not about to bring forward fine differences between orthodox differential minima, but coarse facts easily verified by a few observations.

It is necessary that I should describe and thus expose to criticism the method by which my results have been obtained, for it is not only impossible to obtain fine results by a coarse method, but it is possible to be grossly deceived unless that coarse method is surrounded by some simple precautions. The circumstances and conditions of experiment must be uniform, *i.e.*, we must keep to one set of muscles, we must effect equal lifts at equal intervals, the cutaneous surface of pressure must be maintained unaltered in successive lifts, and not subjected to alterations of temperature; the ordinary manner of "weighing" an object in the hand by jerking it up and down, and thus getting all available sensation by active and by passive pull and pressure, is liable to vary, and should therefore be rejected; above all the expectation of the observee must be as far as possible eliminated by irregularities in the kind and order of application of increments and decrements, and by the avoidance of collateral indications.

The observee is of course blindfolded and his ears stopped by cotton wool, yet it easy enough to unwittingly give him some indication of an alteration made, and he will then "feel" the difference that he expects; we may equally easily prove this influence by giving misleading indications, when he will "feel" differences which do not exist.

After a few preliminary trials a balance method was adopted, viz., a wooden lathe moving round a horizontal axis; a wooden handle half fixed, half moveable, the latter suspended from the former and suspending the lever, a fixed weight of lead to make up 1,000 grammes, a moveable weight 50 of grammes (or more), a small air cushion to support the lathe and prevent shock or sound; the lever is of course graduated for the moveable weight. The muscles tested are the flexors of the fingers, which raise the lower or moveable half of the lever. The lever and weight are supported by the air cushion when the muscles are not in action. The hand rests on the upper half of the handle, the fingers are slightly flexed voluntarily, just touching the lower half of the handle. Duration of lift and interval between successive pairs of lifts are regulated by clock, and are kept as uniform as possible.

The specific question towards which these preparations were directed, was a comparison of weight discrimination (*a*) by voluntary contraction, (*b*) by galvanic excitation, (*c*) by faradic excitation. The observations were undertaken without any distrust of the results ascribed to Bernhardt, or of those described by Ferrier, and relied upon by Bastian, but because the accepted data appeared to me the most direct existing evidence in favour of the exclusively peripheral origin of a muscular sense, and to be the fact most destructive of a central or centro-peripheral theory. I expected, at best, by careful observation to find a moderate difference in the delicacy of discrimination in the three cases, such as might conceivably have escaped observation by rough experiment, and made my preparations accordingly. The observations were not led by undue expectation of results, and the results antedated a critical examination of the positive as distinguished from the dialectical value of the experimental data in question.

Briefly, the results were these. With voluntary contraction of the digital flexors, I distinguished "with certainty,"[1] 1000 and 1150 grammes; with contraction excited by kathodic excitation of the median nerve I distinguished with equal certainty 1,000 and 3000 grammes, or 500 and 1,500 grammes; with contraction excited by faradisation of the median nerve, or of the flexor muscles in the forearm, I am unable to quote any numbers indicative of weights discriminated otherwise than by collateral sources of information; I could sometimes distinguish between one and three kilos., or one and four kilos., but to do so it was necessary to vary the strength of current, and then it was usually found that the distinction was made because the smaller weight was lifted while the larger was not lifted.

These are coarse differences; my first impression was that they proved too much, and were therefore open to suspicion; we shall return to the point in a moment, when we have satisfied the suspicions which these coarse results throw upon the accepted data of Bernhardt and of Ferrier.

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[1] The value to be attached to this expression is discussed above; in my case it signifies about nine times out of ten.

	Discrimination of Weights by Voluntary Contraction.			Discrimination of Weights by Artificial (Galvanic) Contraction.		
	Question.		Answer.	Question.		Answer.
	P.	P + D.		P.	P + D.	
A.D.W.	1000	1100	no diff.	500	1000	no diff.
	,,	1175	heavier	,,	2000	heavier
	,,	1150	heavier	,,	1000	no diff.
	,,	1125	no diff.	,,	1500	heavier
	,,	1200	heavier	,,	1500	heavier
	,,	1100	no diff.	,,	1000	heavier
	,,	1150	heavier?	,,	1000	no diff.
	,,	1150	heavier	,,	1500	heavier
	,,	1125	no diff.	,,	1250	heavier
	,,	1125	heavier?	,,	1000	no diff.
J.L.S.	1000	1050	no diff.	500	1500	no diff.
	,,	1100	heavier	,,	2000	heavier
	,,	1050	no diff.	,,	1000	heavier
	,,	1075	heavier	,,	2000	heavier
	,,	925	lighter	,,	1000	no diff.
	,,	925	no diff.	,,	1000	no diff.
	,,	1075	heavier	,,	1250	heavier
	,,	1100	no diff.	,,	1250	no diff.
	,,	1100	heavier	,,	1250	no diff.
	,,	1075	lighter	,,	2000	heavier

Bernhardt's data have become greatly distorted by quotation. So far from it having been clearly established by Bernhardt that weights "*are as delicately discriminated when muscles are called into play by faradisation as when they are voluntarily contracted*," he says, that in his own case his weight discrimination was "*nearly as delicate*" with muscular faradisation as with voluntary contraction, while in that of a friend, Dr. S——, with ordinary discrimination by voluntary movement, "*no discrimination was possible by muscular faradisation.*" The conclusion which he draws from these premises, conjoined with clinical data, is diametrically opposite to that which is usually ascribed to him, and should therefore be given verbatim.

With the anterior tibial muscles, Bernhardt[1] could by voluntary contraction distinguish with certainty 3 Loth from 0 (? 48 grammes from 0), and 5 Loth added to 1 Pf. (? 80 grms. added to 500); by Faradisation 5 Loth from 0 (? 80 grms. from 0), and ¼ added to 1 Pf. (125 added to 500 grms.) Dr. S—— by voluntary contraction could distinguish 5 Loth from 0 (80 grms. from 0) and 3 to 5 Loth added to ½ Pf. (48 to 80 grms. added to 250); *by Faradisation he could not distinguish* 1 *Pf., or even* 2 *Pf. from* 0 (500, 1000 grms. from 0). Bernhardt says further that with voluntary contraction of the middle finger he could distinguish with certainity 5 Quentchen from 0 (? 20 grms. from 0), and that healthy people can discriminate as finely by electrical means, but that in this case it is by cutaneous sensation. His conclusions (which quite agree with these data as far as they go) are that :—

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[1] The weight units employed by Bernhardt formerly had various values in various parts of Germany; it is assumed above that 1 Pfund = 500 grammes; 1 Loth = 16 grammes; 1 Quentchen = 4 grms.

> Der Muskel selbst also, empfindungslos, mit keinerlei besonderen Apparaten ausgestattet, hat keinen eigenen Sinn, es giebt keinen Muskelsinn, keinen Kraftsinn in der Bedeutung dass specifische Apparate in der Muskelsubstanz uns von deren Wirksamkeit unterrichten.
>
> Der Kraftsinn endlich ist eine Function der Seele, vielleicht unterstützt durch die sensiblem Apparate, welche durch die Kraftäusserung des Muskels irgend wie beeinflusst und gereizt worden sind, aber nicht ihm sondern seiner Umgebung angehören.

Bernhardt cannot therefore be appealed to as a witness in favour of the exclusively peripheral theory, and I think we must reject even the very moderate value at which they have been rated in this respect by Professor James.[1]

It is no very hazarded conclusion from these several premises to say that the nominal or dialectical value at which these "experimental" data have been estimated is in excess of their real or positive value.

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[1] Bernhardt has shown in a rough experimental way that our estimation is as delicately graduated when our wills are passive and our muscles made to contract by direct faradization, as when we ourselves innervate them ("Principles of Psychology," Vol. II., p. 502).—Dr. Ferrier ("Functions of the Brain," *p.* 392) says:—"Experiments made in reference to this point by myself, with the assistance of Dr. Lauder-Brunton, gave such results as clearly to indicate the retention of muscular discrimination when the muscles were excited to contract by the galvanic current. The method I adopted was to determine, blindfolded, in the first instance, the differences in weight which could be discriminated by my hand held flat on a cushion, and then to test the muscular discrimination of the same weight when the wrist was flexed so as to raise the weight with the fingers. By repeated experiments with weights varying from one to six ounces the average discrimination by the sense of cutaneous pressure was found to be about one-third, while the muscular discrimination accorded pretty nearly with the 1-17th, as usually found to be the rule.

"The same experiments were then made with the same hand as regards

With regard to the actual question in hand, it is advisable to recognise what conclusion the facts I have asserted do not bear. The great inferiority of weight discrimination by peripheral as compared with central excitation is simply a direct negative of the experimental premise upon which Dr. Bastian relies, but it cannot itself be utilised as a positive premise contributing to the conclusion that the sense of effort is not wholly peripheral, but partly or wholly central. The numbers given above prove too much. With galvanic excitation 1,500 grammes is distinguished from 500; 1,000 grammes and 500 grammes are usually not distinguished or distinguished wrong. But we know that by mere skin pressure—which is assuredly a stimulus of purely peripheral origin, 1,000 grammes and 500 grammes are infallibly distinguished; it is evident that skin pressure comes into play in the lift of a weight by direct muscular excitation, and it is at first sight somewhat surprising to find the discrimination less delicate in the latter case than by simple pressure. We may however probably easily enough account for the fact; the excitation may cause sensation inhibiting other sensation, or it may block centripetal impulses along nerves. The point need not be pursued; enough has been said to show that while such experiments are valuable *against* the conclusion that the sense of contraction is wholly peripheral, they are in themselves of no value *for* the conclusion that they are not wholly peripheral. This last conclusion rests, however, upon other premises.

Another datum has been invoked, which as a *datum scriptum* is of great weight, but on examination is found to be exceedingly unsubstantial. The observations of Sachs are frequently referred to as having demonstrated anatomi-

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cutaneous pressure, and by galvanic excitation of the flexor muscles of the hand, so applied as to cause repeated raising of the weight by the fingers. Again the sense of pressure averaged the normal, and again muscular discrimination was found to be almost as accurate as in the former experiments, when the raising of the fingers depended on voluntary effort.

"Cutaneous pressure being thus allowed for in both cases, the muscular discrimination by means of the centripetal impressions generated by muscular contraction alone, not depending on voluntary motor impulse, is clearly established."

cally and physiologically the sensory nerves of muscle, and the demonstration is appealed to as a firm point of departure. The actual facts are these:—Sachs examined the nerve of the sartorius muscle of the frog some time after the section of the anterior roots, and amid a majority of degenerated (motor) fibres found two undegenerated fibres; he inferred that these were fibres belonging to the posterior root and therefore sensory. He made the counter experiment by searching for degenerated fibres some time after section of the posterior root and failed; he attributes this failure to preservation of his two sensory fibres by sensory impulses; he attempted to verify the opinion and failed. This is the anatomical demonstration.

Sachs' physiological demonstration is as follows:—Strychninised frogs were used, the sartorius and its nerve were isolated and separately excited by induction shocks, reflex contractions were obtained.

It seems to me that this is a very narrow and insecure basis, both anatomically and physiologically, upon which to rest the dogma of sensory muscular nerves.[1]

But it may be said that by sensory nerves of muscle are meant the nerves of their connective tissue accessories, especially of their tendons, of which the very evident nerve-endings must be sensory since they cannot be motor. This may be freely admitted, and in the conclusions it will correspondingly be found that a peripheral *contribution* to a sense of contraction, postulating sensory nerves from the motor organs is recognised to exist. We need not, however, confuse the muscular and tendinous afferent nerves—the latter have been proved to exist, the former have not; and if they had it would not affect our conclusion.[2]

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[1] A very similar appreciation of Sachs' data has been given by Mays.—*Ztsch. F. Biol.*, vol. xx.

[2] The observations recorded by Professor Chauveau in his paper on the "Nervous Circuit," postulate the existence of muscular *sensory* nerves, and considerably strengthen the evidence of their existence. The purport of the remarks made in the text is strictly as follows:—The existence of muscular sensory nerves is not established on sufficiently positive grounds for it to be admissible as a datum in support of the doctrine of kinæsthesis.

It is indeed conceded in the conclusion of this paper that passive muscular effects probably *contribute* to our knowledge of movement; this postulates of

Up to this point we have been on physiological ground, and we have not there seen any cogent proof of the exclusively peripheral character of the sense of contraction or muscular sense. But the central initial origin of the motor feeling—Wundt's Innervationsgefühl—has also been debated upon clinical grounds, and from a purely psychological standpoint—and these cannot properly be left without mention.

I do not feel competent to deal with clinical data with any degree of assurance, as I possess no first-hand information on the subject, and am often in considerable doubt whether "loss of muscular sense" or "preservation of muscular sense" are verbal formulæ or duly authenticated facts. Indeed I cannot, otherwise than by the supposition that the expression "muscular sense" denotes very various objective phenomena, understand the flagrant and fundamental contradictions dogmatically enunciated by the highest clinical authorities. On the one hand we have the statements of Leyden, of Bastian and of many others, that an ataxy, assumed to be due to a loss of muscular sense, may exist without diminution of tactile and common sensibility. On the other hand Ferrier's emphatic statements that "the same condition which abolishes cutaneous sensibility, also entirely annihilates the so-called muscular sense. Loss of the muscular sense never occurs without general anæsthesia of the limb (p. 380). There does not appear to be a single fact which would indicate that the muscular sense can be abolished and the other forms of sensibility of the limb continue."

course the existence of muscular sensory nerves. But it is one thing for a physiological argument to lead up to muscular sensory nerves, it is another for such an argument to lead off from them. In the first case the logical direction is from the more known to the less known, in the second it is from the less known to the more known.

That the anatomical reality of muscular sensory nerves is most unassured was first indicated by Waller's well-known observations on the distribution of degenerated fibres after section of the posterior roots through which all afferent fibres pass. The passage is short enough to be quoted verbatim; it refers to the state of muscular and cutaneous nerves of the frog after section of the posterior roots. "Sur un animal qui avait survécu un mois et demi à cette opération, j'ai trouvé tous les nerfs de la peau désorganisés, et la substance intra-tubulaire au deuxième degré d'altération. . . . Dans les muscles, les fibres nerveuses me paraissaient toutes normales, et je n'ai pu apercevoir aucune trace dans les ramifications ultimes de fibres désorganisées." —(Waller, *Nouvelle Méthode Anatomique*, 1851).

Then to complete the clinical picture we have Bastian showing us that "loss of muscular sense sometimes accompanies hemiplegia," and Ferrier "that loss of muscular sense is not associated with hemiplegia."

It appears to me (1) that there is a radical difference of opinion between clinical experts as to the fundamental clinical facts, which are presented as black or white according to the doctrinal point of view from which they are regarded ; (2) that the clinical facts even if proved in one or other form, remain "lump-facts" which afford no isolated premise to be utilised in the main question we are dealing with, *re* the sensations which accompany and follow muscular action, viz., are they peripheral or are they central, or are they peripheral *and* central?

It is therefore only to avoid the reproach of not paying sufficient heed to "obvious clinical facts" that I have alluded to the clinical argument at all, which, in my opinion, has not proved whether there is or is not a "muscular sense," nor what are the essential signs of its presence or of its loss, nor what is the physiological origin and centre of the endowment. "Experiments" on hysterical hemianæsthetics, *e.g.* (the much-quoted case of Demeaux studied in 1840) will hardly be relied upon in 1891 as positive evidence on either side.

The psychological standpoint.—The reluctance with which I embarked upon muscular sense considerations reached its maximum when the purely psychological presentation of the subject had to be faced. The case as presented by psychologists, is made to turn mainly upon doctrinal arguments and subjective pleadings, which although usually characterised as "cogent," do not, in my opinion, force the verdict in one sense or another. On the contrary the psychological arguments for and against this or that view, seem to me to obscure the real and simple point at issue, viz., whether the sensation due to movement arises from material changes in the centre or in the periphery or in both situations. This may justly be characterised as a sweeping expression of opinion, and I therefore hasten to add that it is only advanced as an expression of opinion, and that among the large mass of literature bearing upon the point, the very able and comprehensive summary recently given by Professor James (*Principles of Psychology*, Vol. II.) has more particularly contributed to the formation of that opinion. "Perhaps," "It may be," are the comments aroused by his positive or constructive arguments; "Certainly," "Of course," those aroused by his negative or destructive arguments. This being so, I am most unwilling to add to the case by the pleading of one set of probabilities as opposed to another. I prefer simply to requote a passage from an earlier writing by the same author, which has been endorsed as most weighty by one of the most authoritative believers in the pure peripheral theory; this will form the fairest presentation of the kind of argument which is offered as cogent, but which cannot but be characterised as wholly inconclusive, inasmuch as it elicits the comment "Perhaps," but not the comment "Certainly." The passage is a requotation of James as quoted by Ferrier (Functions of the Brain, second edit., p. 382):—

No one has put this[1] more clearly than Professor W. James ("The Feeling of Effort," *Anniversary Memoirs of the Boston Society of Natural History*, Boston, 1880), and his remarks are worthy of quotation *in extenso*. "Now what can be gained by the interposition of this second relay of feeling between the idea and the movement? Nothing on the score of economy of nerve tracts; for it takes just as many of them to associate a million ideas with a million motor feelings, each specific, as to associate the same million ideas with a million insentient motor centres. And nothing on the score of precision; for the only conceivable way in which they might further precision would be by giving to a mind whose notion of the end was vague a sort of halting stage with sharper imagery on which to collect its wits before uttering its *fiat*. But not only are the conscious discriminations between "ends" much sharper than anyone pretends the shades of difference between feelings of innervation to be, but even were this not the case, it is impossible to see how a mind with its end vaguely conceived could tell out of a lot of *Innervationsgefühle*, were they never so sharply differentiated, which one fitted that end exactly, and which did not. A sharply conceived end will, on the other hand, directly awaken a distinct movement as easily as it will awaken a distinct feeling of innervation. If feelings can go astray through vagueness, surely the fewer steps of feeling there are interposed, the more securely we shall act. We ought, then, on *a priori* ground alone to regard the *Innervationsgefühl* as a pure encumbrance.

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[1] *i.e.*, the superiority of the peripheral as compared with the central theory of the feeling of effort.

"Let us turn now to *a posteriori* evidence. It is a notorious fact, recognised by all writers on voluntary motion, that the will seems concerned only with results, and not with the muscular details by which they are executed. But when we say 'results' what is it exactly that we mean? We mean, of course, the movements objectively considered, and revealing themselves (as either accomplished or in process of being accomplished), to our sensible perceptions. Our idea, notion, thought, of a movement, what we *mean* whenever we speak of the movement, is this sensible perception which we get of it when it is taking place or has completely occurred. What, then, is this sensible perception? What does it introspectively seem to be?

"I unhesitatingly answer: an aggregate of *afferent* feelings, coming primarily from the contraction of the muscles, the stretching of tendons, ligaments, and skin, and the rubbing and pressing of joints; and secondarily, from the eye, the ear, the skin, nose, or palate, any or all of which may be indirectly affected by the movement as it takes place in another part of the body. The only idea of a movement which we *can* possess is composed of images of these its afferent effects. By these differences alone are movements mentally distinguished from each other, and these differences are sufficient for all the discriminations we can possibly need to make when we intend one movement rather than another."

The view-point from which I have regarded the matter does not permit me to see the cogency of the above argument. I cannot, however, controvert it, nor can I controvert the more lengthy developments which have been given to it by Professor James in his *Principles of Psychology* (Vol. II., p. 449 to p. 518). In fact, I fully admit the *destructive* character of his arguments as opposed to the introspective and circumstantial evidence of a central feeling of innervation—evidence which has appeared to me to be as little cogent as the subjective and "experimental" evidence which is advanced in support of the purely peripheral doctrine. It is the constructive character of the argument that appears to me to be illusory.

Still, although I cannot follow Professor James step by step on the slippery ground of the above quotation and of much of his more recent pleading, I feel that the most elementary courtesy makes it necessary that I should not merely assert that a subjective view which I cannot accept appears to me as an unreal phenomenal picture, but that I should go a step further and expose as clearly as possible to a similar adverse opinion my own subjective views on the same topic. This is formally done in the following paragraph, of which I am well aware that the subjective element forms a principal ingredient.

§16—Hypothetical Considerations and General Conclusions

Fully recognising the distinction which should be maintained between fact and hypothesis, yet that *both* elements are as essential to living knowledge as male and female elements are to living matter, it appears to me that in the hypothetical relations between central and peripheral phenomena in the general government of muscular action, we must distribute the shares of function between centre and periphery otherwise than is usually done.

The very general disposition of opinion is towards the admission of reflex response to a muscular sense, as playing a preponderant if not exclusive part in the *minute* government of the body.[1] No doubt we are informed of the position of our limbs partly by the state of the skin, partly by the state of the muscles, and admitting that the tendon of muscle (*perhaps* even the muscle itself) possesses afferent nerves, we are tempted to assume that the sense of effort which we feel is entirely obtained through centripetal channels, *i.e.*, that we have a "muscular sense." We estimate weight and difference of weight chiefly by means of trial efforts by which we ascertain how much our muscles must be contracted in order to lift the weights, or to arrest their descent, and we may reasonably suppose that the amount of contraction is estimated by a "muscular sense." But this property in the form of centripetal process *from muscle* is not supported by any direct proof, and so long as the alternative hypothesis of expended energy in "motor" centres is not disproved, it is not possible to admit that the feeling is entirely of peripheral origin, nor that the muscular contribution is the predominant factor among its peripheral constituents. I am not denying that it is *a* factor, co-operative with cutaneous and articular[1] factors, as well as with visual and labyrinthine guidance, but I am denying that it has been proved to be a factor, and pleading against the exclusion of the central factor.

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[1] This position is very forcibly put in an account of "sensomotor" actions recently given by Exner in Pflüger's Archiv., vol. 48, p. 592, 1891.

On the minutely reflex view the muscular fibres at the periphery form an army, the units of which are not only constantly "in hand," subject to cerebral control in response to true sensation, and constantly "in hand" subject to reflex spinal control, but they also never act otherwise than indirectly. Leaving on one side volitional control, to concentrate our attention upon the spinal and muscular relation, we find ourselves urged to imagine that, *e.g.*, the so-called automatic movements of locomotion and of equilibration are item by item reflex contractions responsive to extensile excitations.

This may be acceptable as regards the slow large movements of massive groups of muscle, but how slow and cumbersome this arrangement appears when we consider the co-ordinated modulations of small rapid movements of little muscles. We can imagine the individual movements of equilibration to be reflex, they are slow enough; we cannot imagine the individual movements of vocalisation to be reflex; they are much too rapid. And in this second case we are constrained to admit that the constituent items in the co-ordinate series are measured out in due proportion and sequence in the emissive centre, independently of discrete peripheral calls through "muscular sense." Why not also in the first case? No doubt true reflex adjustments are continually ebbing and flowing in the maintenance and alteration of muscular attitudes, and we make the coarse adjustments of our muscles in response to what we feel from the periphery by all our organs, and perhaps more particularly by our muscles and ligaments and cartilages. But we cannot imagine that the fine modulations and rapid adjustments occur in such a roundabout way. They must be rightly measured out at their source of origin and it is probable that they may also occur at the periphery in direct response to extensile stimuli. This supposition brings us to an objective phenomenon which we can measure, and thereby obtain what I think to be proof positive of a direct peripheral regulation, such as we have suggested to us by *a priori* considerations. I refer to the much debated and hackneyed phenomenon, called tendon reflex, of which the lost time is incompatibly short as compared with the shortest known reflexes, and practically indistinguishable from a direct latency.

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[1] For the articular element in the muscular sense, Cf. Goldscheider's papers in Du Bois-Reymond's Archiv. 1889, p. 369, and suppl. p. 141.

I entirely accept the existence of centripetal impulses arising from resistance to motion, caused by varying tension of motor organs—and find sufficient grounds for the admission in definite experimental evidence. But I deny the exclusive part assigned to reflex control by this means, in conjunction with visual, labyrinthine and cutaneous guidance, and plead for the admission of a contributory control by the central sense of motor innervation, and of a further contributory control by immediate peripheral response to alterations of muscular tension—the direct "responsibility" of the muscle itself.[1]

The main objective evidence offered in proof of the two pleas is (1) the demonstrable fact that fatigue, the consequence of effort, manifests central as well as peripheral objective signs. (2) The demonstrable fact that peripheral response to extensile vibration can occur so rapidly that reference to and from the spinal axis cannot have taken place.

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[1] I have used this word literally, in spite of its connotations, and for this reason, that isolated muscle has properties which if they were witnessed in an organism would be regarded as signs of intelligence. Muscle is an intelligent agent rather than an inert instrument the trigger of which is pulled. It evolves much or little heat, according as it pulls against much or little resistance (Heidenhain). The greater the resistance, the greater the heat, the greater the chemical action. If more energy is wanted from muscle, more energy is served up. And, be it remarked, the adaptation is in *isolated* bloodless muscle, independent of nervous system. It is as if we had rifles shooting hard and soft, according to distance, measuring out energy in proportion to requirement.

In sum the view which I am inclined to take is that the duly co-ordinate action of the muscular executive depends upon three factors.

1. Upon co-ordination at the central origin of action, which co-ordination is the resultant of past experience, and consists in the emission of nerve-impulses grouped and measured in conformity with intended and foreseen effects.

2. Upon appropriate and corrective reflex response, of which the immediate causes or stimuli are centripetal impulses from sense organs, inclusive of those less obvious impulses from the skin, muscles, and articulations, which are collectively referred to a muscular sense.

3. Upon the direct muscular response to passive stretching and extensile vibrations.

With regard to the incidence of fatigue phenomena, considered as representing in intensified form the incidence of changes which underlie "the muscular sense," and the "sense of effort," the conclusions which appear to me of most interest are—

1. That the conducting channels (nerve-fibres) do not suffer fatigue, but that fatigue, consequent upon excessive nervous action, whether voluntary or excited, especially involves the terminal or junctional organs in the cerebro-muscular chain.

2. That in voluntary fatigue the degree of change is in decreasing ratio from centre to periphery. In other words that the cell of higher function is, relatively to the amount of effect which it can produce, more exhaustible than the cell which is subordinate to it in the cerebro-muscular chain. So that central fatigue is protective from peripheral fatigue; and although normally in the body the dynamic effect of the physiological stimulus emitted by a "motor" centre far exceeds the maximal effect which can be elicited by direct experimental excitation, yet that physiological stimulus, by

virtue of the rapid exhaustibility of the organ from which it proceeds, cannot normally "overdrive" and exhaust the subordinate elements of the motor apparatus.

As being perfectly conformable with this view I may also point to the most familiar of all the objective phenomena of fatigue—normal sleep. The brain sleeps, but sleep is not an exclusive attribute of the brain, it extends in less degree to the subordinate nerve-centres, and is attended with a diminished activity of the periphery itself—as evidenced in slower respiration, slower pulse, diminished excretion of carbonic acid and of urine. It is obvious that the degree of this negative process is greater at the centre than at the periphery, *i.e.*, in a decreasing ratio from centre to periphery, and we may recognise in the varying depths of sleep —from its lightest or "cerebral" forms to its deepest cerebro-spinal manifestations—the law of central maximum and centrifugal decrease which is characteristic of fatigue. "In deep sleep consciousness is absent, stimuli of even considerable strength may be insufficient to arouse sensation, the muscles are completely relaxed, the so-called tendon-reflexes are in abeyance, the deep sleeper lies dreamless and motionless. All these signs are wanting in sleep which is light or partial: consciousness is blunted, but not absent; ideas string themselves together in more or less congruous sequence and form dreams, or are aroused by more or less appropriate stimuli, or they actually arouse movements more or less co-ordinate and purposive, such as are seen in their fullest expression in the actions of sleep-talkers and of sleep-walkers."

The connection between this mode of viewing the incidence of fatigue, and the opinion we may hold with regard to the seat of production of those psychophysical changes underlying sensations of movement and of effort is —admitting the relationship urged in § 4—to the following effect:—

Fatigue phenomena are manifested in the entire series of organs from centre to periphery in consequence of voluntary exertion, therefore material changes are produced throughout the series *with* voluntary exertion. We have seen independent reasons for the admission that co-ordinated muscular movements depend upon precisely modulated changes in all the knots of the centro-muscular organ, and that admission is at least highly suggestive of the simple conception of a series of excitable clumps, all of which are altered by activity, and all of which are reviewed in consciousness. If we form such a conception, we shall not say with Bastian that the physical change felt as "sense of effort" is peripheral, nor, with Bain, that it is central, but, with Wundt, that it is central and peripheral.

I also consider that the centrifugal law of fatigue studied in insulative experiments, and representative of molecular alterations associated with motor activity, is a part of a first principle in medical philosophy established by clinical observation—the Jacksonian law of lability. The lower function is deep rooted, the higher function is new grown and growing. The organic foundation is firm, the organic crown is mobile; expenditure in each single movement, the expenditure of a day's work, the expenditure of a lifetime, the accelerated expenditure of the over-willing or over-mastered slave, the narcotic after-effect of the stimulation effect, the accidental episode of epilepsy, have as their common feature, a dissolution of matter beginning at the crown of the nervous edifice, and deepening in ever smaller ratio throughout its lower strata. Last to come is first to go, first to go is most to go. First to come is last to go, last to go is least to go.

POSTSCRIPT. Reviewing the subject matter of this paper after a lapse of three months it appears to me possible that a hurried reader may miss its main neurological purport.

The points upon which most stress is laid are:—

1. The utilisation of a study of the incidence of fatigue *after-effects* as a guide to the incidence of motor *effects*.

2. The affirmation of the statements that voluntary fatigue is in major part central, in minor part muscular; that central fatigue is protective from peripheral fatigue.

3. The demonstration of the varying relations between muscular swelling and muscular shortening in voluntary contraction; the identification of a *prolonged* lateral effect as mainly a central phenomenon similiar to the "contracture" of neurologists, and of an *increased* lateral effect (relatively to the simultaneous longitudinal effect) as mainly a peripheral phenomenon similar to the "contracture" of physiologists.

4. The contradiction of the statement that weight-discrimination is as delicate by directly excited contraction as by voluntary contraction.

The points upon which less stress is laid, although they have of necessity received attention, are:—

1. The experimental verification of the end-plate effect in human muscle.
2. The experimental determination of the source or cause of the increased effort effect in voluntary fatigue, and of the prolongation of the dynamic effect in muscle.

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HUGHLINGS-JACKSON ON THE CONNECTION BETWEEN THE MIND AND THE BRAIN.

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Running through the later writings of Hughlings-Jackson, or at least through such of them as deal with the more abstract problems in nervous pathology, will be found a very decided opinion regarding the relation between the mind and the nervous system. This distinguished neurologist has consistently and repeatedly maintained a view regarding this relationship which has occupied a more or less prominent place in his writings. It is true that this theme has not, so far as the writer knows, ever been presented as the main topic under discussion, yet it has been emphasised so often, and in such decided language that, considering Hughlings-Jackson's standing as a neurologist, it is desirable that it should not be allowed to pass without careful examination.

A passage wherein this view has been as distinctly expressed as anywhere else, is the following: "Now, I speak of the relation of consciousness to nervous states. The doctrine I hold is, first, that states of consciousness (or synonymously, states of mind), are utterly different from nervous states; second, that the two things occur together—that for every mental state there is a correlative nervous state; third, that although the two things occur in parallelism, there is no interference of one with the other. This may be called the doctrine of concomitance. Thus in the case of visual perception, there is an unbroken physical circuit, complete reflex action, from sensory periphery through highest centres back to muscular periphery. The visual image, a purely mental state, occurs in parallelism with—*arises during* (not *from*)—the activities of the two highest links of this purely physical chain—so to speak, it ‘stands outside’ these links.

“It seems to me that the doctrine of concomitance is at any rate convenient in the study of nervous diseases. It, or an essentially similar doctrine, is held by Hamilton, J. S. Mill, Clifford, Spencer, Max Müller, Bain, Huxley, Du Bois Reymond, Laycock, Tyndall, Hermann, and David Ferrier.

“Those who accept the doctrine of concomitance do not believe that volitions, ideas, and emotions produce movements or any other physical states. They would not say that an hysterical woman did not do this or that because she lacked will; that an aphasic did not speak because he had lost the memory of words, and that a comatose patient did not move because he had lost consciousness. On the contrary, they would give, or try to find, materialistic explanations of physical inabilities. I do not try to show what is the nature of the relation between mental and nervous states.”[1]

Again: “We must be very careful both in ophthalmological and neurological studies not to confound sensations—colours for an example, which are states of mind—with activities of sensory elements, which are states of body. We must not speak of any mental states as occurring *from*, but as arising *during* discharges; only physical effects, such as movements, arise *from* nervous discharges.”[2]

From these quotations it will be seen that Hughlings-Jackson sharply differentiates the psychical from the physical, and widens the chasm between them to proportions as great as it ever attained in the past. He lends the authority of his name to what may be called the popular opinion. If this opinion be correct, all well and good; but if it be erroneous—as many competent to judge believe—it is time that this statement be challenged.

I hope to be able to show in the course of these pages that this doctrine of “concomitance,” as just expressed, is one that leads to consequences more fallacious than even the most materialistic, which are bad enough.

[1] “Evolution and Dissolution of the Nervous System.”

[2] “Ophthalmology and Diseases of the Nervous System.”

I cannot help suspecting that Hughlings-Jackson has not completely grasped the full meaning of this problem. To group together Hamilton, Clifford, Spencer, Bain, Huxley, and Tyndall, to say nothing of the others, as holding essentially the same doctrine, is to my mind much the same thing as putting Salisbury, Gladstone, Chamberlain, and Labouchere into one political boat, and saying they hold essentially the same opinions. If one does not see the essential difference between the opinions of Clifford and Huxley, one can scarcely have a clear idea of the matter.

Not the least of Hughlings-Jackson's sins is the seduction of his pupil, Mercier,[1] who has even more vehemently expressed himself to the same effect, though both claim the authority of Spencer for their opinions. But anyone familiar with Spencer will hardly hold him responsible for such errors. Mercier writes, "Try to think of a feeling passing along a nerve. . . . The axis cylinders are grey threads of proteine substance, which is made up, like all other matter, of molecules swinging in space. Now, where is pain? Is it in the molecules or in the intervening space? And how does it pass along the nerve? Does it jump from molecule to molecule, or does it flow in the interstices? If the former, pain must be solid; if the latter, it must be a fluid, both of which hypotheses are manifestly nonsense. . . . But it may be said, Pain, we know, is not really in the nerves, it is in the brain. Again the same problem awaits us: the brain is made of cells and fibres. Is pain in the cells? Is it in the fibres?" &c., &c. "Let us try to imagine," he continues, "an idea of food producing a movement—say, of carrying food to the mouth. . . . Try to imagine the idea of a beefsteak binding two molecules together. It is impossible. Equally impossible is it to imagine a similar idea loosening the attractive force between two molecules."

I agree with Mercier that such hypotheses are "manifestly nonsense;" but they are nonsense because of the nonsense connoted by the terms matter and mind as employed by Mercier. But notwithstanding that we (who hold the views which will be presently explained) are in full accord with him on this point, nevertheless we maintain that Hughlings-Jackson and Mercier are at fault in allowing themselves to be "driven to the conclusion that pain and matter are things with no community in nature, are facts of totally different orders, and cannot be reduced to any common term." Still more, our contention is that no one could seriously propose the question, "Can the idea of a beef-steak bind two molecules together?" as one at all involved in the problem, without having a total misconception of the nature of the subject under discussion; no one could ask such a question and possess a correct idea of the nature of matter. These extraordinary hypotheses are only relevant on the supposition that matter, that molecules and motion, have objective reality, as we know them, and that they are not merely states of consciousness. I can imagine the horror with which Spencer would contemplate such notions being imputed to him after all he has written to discredit them.

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[1] "The Nervous System and the Mind."

But there is one very serious objection to Hughlings-Jackson's views, which may as well be stated here. "Those who accept the doctrine of concomitance," he naïvely remarks, as if it were no objection to his hypothesis, "do not believe that volitions, ideas and emotions produce movements, or any other physical states." This is the position taken by Huxley, who once said (whether he still maintains it I do not know), "The consciousness of brutes [and men] would appear to be related to the mechanism of their body simply as a collateral product of its working, and to be as completely without the power of modifying that working as the steam whistle, which accompanies the work of a locomotive engine, is without influence upon its machinery. . . . . And to take an extreme illustration, the feeling we call volition is not the cause of a voluntary act, but the symbol of that state of the brain which is the immediate cause of that act."

Mercier with much emphasis adopts, if I interpret him correctly, the same "steam-whistle" opinion, though expressing himself in different language.

And they are right so far as this conclusion is a logical consequence of their premises. It is the inevitable conclusion to which they must be driven. If mind is something that stands entirely apart from molecules and motion, if it has only a parallel existence with cerebral processes, it cannot enter as a link in the chain of events, and consciousness can have nothing to do with governing our actions. We are nothing, as Huxley has said, but automata.

Now I do not wish to speak except with the highest deference for those who hold opposing views. I know how easy it is for the mind to deceive itself in matters of this kind; how difficult it is to free one's self from the ideas which by long habit are connoted by language, and which consequently prevent our viewing a thing from a new aspect. But I do wish to emphasise the fact that any doctrine which ultimately leads to denial of volition as a cause of action is, as Mercier would say, "nonsense," and doomed to failure. If one is moved to sympathy at the misery of a beggar, and following one's sympathy one gives a dollar to that beggar, the giver is satisfied that his feelings of sympathy—his states of consciousness—directly control his muscular acts and move his fingers to take a dollar bill out of his pocket and give it away. This is a fact of direct experience, and is worth a whole volume of scientific erudition.

If, under the influence of anger, I strike a man, there is little use in my trying to shift the responsibility from my temper to the shoulders of my grey matter, and in my telling the world that my outburst of temper was only a sort of "steam whistle;" that it told me what my right hand was going to do, but had no more to do with the hitting than had that of the judge on the bench who is going to try my case.

Popular language correctly expresses the facts in such cases, and any scientific doctrine which attempts to explain the relation of mind to body, and does not recognise this truth, will never be accepted by common-sense people. The contention of those who hold the doctrine which will be developed later, is that the "steam-whistle" advocates have been logically driven to their conclusion; but the reason for this is that though their logic is faultless, their premises are wrong, the conditions of the problem not being thoroughly understood. Well-known truths regarding the nature of so-called "matter" have, in practice, been neglected, or their full bearing on the question been overlooked. That there is a solution of this question, which on the one hand does not disregard these truths, and on the other hand recognises volition as a cause of muscular action, we believe. This solution thus far for the most part has been neglected; but the reason for this is plainly because it has not been understood, and not because any serious objection has been urged against its final conclusions.

This matter is not one which purely concerns the psychologist, with whom it has hitherto been customary to leave it as a question of metaphysics. It concerns as much the alienist and neurologist, and even the general practising physician, with all of whom it is a matter of real practical importance. How can one understand, for example, how a psychical shock, such as would be incurred in a railway disaster, can produce not only mental perturbations, but disturbances of the various physiological functions of the body, known as somatic? How can one really understand how such a shock could cause disturbances of the heart, the viscera, neuralgic pain in various parts of the body, tumour, vaso-motor disturbances, spasms, and what not, unless one has a clear conception of the relation between mental action and cerebral action? How understand the true genesis of neuro-mimesis and hysteria, with all their protean manifestations, or conversely, how can one possess any distinct picture of the mode by which a purely physical concussion, or disturbance of the circulation, causes an impairment of speech, reasoning powers, and other mental faculties, unless he has a definite idea of the relation between these two classes of facts? If we look back to the time when mind was regarded as something apart from the body, and consider the views which obtained in those days regarding the nature of insanity and the barbarous treatment of the insane, which was their natural consequence, we realize what the practical results have been in these later years from obtaining a knowledge of the dependency of a diseased mind on a diseased brain; and if we can penetrate deeper still into the inner recesses of the nervous system, and can extract the secret of the more intimate relation between the mind and the brain matter, we can scarcely doubt that much that is now obscure in the pathology of many neuroses and psychoses would become intelligible. Therefore it is, I say, that this question is a practical one, and one which the neurologist should seek to understand.

The negative view to which Dr. Hughlings-Jackson has repeatedly given expression—that we have no knowledge whatsoever regarding the subject, and that we stand face to face with a great mystery—is that which is popularly held, although amongst those who have thought much on the subject there is a minority whose opinions are entitled to weight, who believe that a satisfactory solution has been found. This solution has not been generally accepted, and indeed I think I keep well inside the limits of moderation in saying that it has not been really understood by one out of every ten even of those who have undertaken to write upon the question. For this reason various false conceptions are usually to be found underlying every discussion of this most perplexing problem. And yet it is this minority view towards which opinion is gradually converging.

That every mental act is accompanied by a physical change in the brain has become an accepted axiom, and yet, having due regard to the accuracy of language, it may be said that *such a statement is absolutely false, and is the basis of most of the fallacious views on the subject.* This may seem paradoxical, and probably will be challenged by every reader who has followed me thus far, and yet it is a fact which I hope to be able to make clear before I have finished.

"Does the mental action follow the physical action, or does the physical action follow the mental?" How do neural vibrations in the brain become transformed into thought and feeling? These are questions which are frequently asked, and the questioner shakes his head and tells us there is no use trying to answer them. The essayist, if he be an essayist, says that these questions are insoluble, and that we are as far as ever from obtaining a solution of the mystery. This is perfectly true. We are as far as ever from doing this, and it is safe to say that we never shall answer these questions, for the simple reason that the questions themselves are mere nonsense. They involve assumptions which are not true, and therefore the questions are absurdities and cannot be answered. We cannot say how brain action is transformed into thought, because it is not "transformed" at all. Nor shall we be able to discover whether mental action follows the physical, or *vice versâ*, because neither follows the other. The erroneous assumption here is that there are two different facts (the mental and the physical), each having a separate and independent existence *in the same individual,* whereas there is in reality only *one fact*, and one thing cannot very well be transformed into or follow itself. That we have two different and separate conceptions—one, the so-called mental facts, the other, the so-called physical facts—is perfectly true; they form a part of common experience. But whether there are two different and separate existences corresponding to these conceptions is another matter, and is something which cannot be left to common experience to determine. For aught that appears on the surface there may be but one real existence, or fact, which is the basis of both conceptions, and, without anticipating further the argument, it may be said that this is the explanation to which the best thought at the present day is tending. I am not referring to the notion that mental and physical facts are but "different aspects of one and the same underlying thing"—a most hazy and confused notion—but to a very different idea. I prefer that this idea shall be developed later as we go on, rather than by expressing it now incur the risk of being misunderstood at the outset before the ground has been prepared. I shall have, then, to ask the reader to temporarily suspend his judgment and to follow me with open-mindedness to the end, and to try to get at my real meaning which I fear my words will but poorly express.

I take it that the reason why this question has escaped a satisfactory explanation is because we have not examined sufficiently deeply into the nature of the facts under investigation, and have not reduced them to their lowest terms. In matters of this kind it is essential that we should first resolve the facts into their component elements and find out what manner of things they are—whether they are what they seem to be on the surface, or something else, before attempting to deal with them. I say in matters of this kind, because in other fields of inquiry—in the physical sciences, for example—it is not essential that we should make this preliminary enquiry into the exact nature of the things whose phenomena we are investigating. If you, who are interested in chemistry, wish to learn the exact amount of sulphuric acid and zinc necessary to produce a given amount of sulphate of zinc, or desire to ascertain the chemical action which takes place in the process; or if I wish to know the laws of electro-magnetism, it is not necessary that either of us should know the exact nature of material bodies, nor that we should have more than a common expert knowledge of chemical substances and of the nature of electricity. Whatever error may exist in our conceptions of these material facts will be the same on both sides of the equation, and may be neglected. It is not even necessary to know the nature of electricity, for whatever it be—whether a fluid, as was formerly held, or whether a mode of motion—it is only its laws that we are investigating; while, underlying these, electricity itself persists as a constant unvarying factor, and any result that may be arrived at will not be affected by any error in our notion of electrical force. We are dealing from beginning to end with facts of the same order or class, which have equivalent values so far as their intrinsic nature is concerned. Likewise in dealing with problems in physiology, if, for example, we wish to determine the relations between a peripheral stimulation and a resultant motor action, it is not necessary to know the essential nature of nerve force or the exact chemical composition of the protoplasm of nerve cells or the true nature of the brain matter, because these are constant factors in the reaction, and bear the same relation to the ingoing stimulation as to the outgoing one. So, too, in dealing with matters of psychology. If we wish to study the laws of memory, or of volition, or of the association of ideas, it is not necessary to go deeply into the nature of mind for similar reasons. Thought, sensations, feelings are all facts of the same order, just as zinc, sulphuric acid, electricity, and heat are all material facts, and therefore of the same class. The one class is psychical, the other class material, and so long as we are only dealing with facts of one class, it is not necessary to reduce them to their lowest terms; but when we wish to ascertain the relation between facts of two different orders — between psychical facts on the one hand and physical facts on the other—to learn if they have anything in common, discover their likenesses or unlikenesses, it becomes necessary to resolve them into their component elements before attempting to compare them. In other words, if x is on both sides of the equation its value may be neglected, but if x is on the one side and y on the other the equation cannot be established until the value of x and y be known.

Pursuing, then, our enquiry along these lines, let us see what is the composition of a psychical state and what that of a cerebral state. It will be, perhaps, more convenient to consider the latter first. When we speak of cerebral activity, it has been agreed that we mean a form of molecular motion —neural vibration it is often called. The exact nature of this motion has not been made out, but there can be no doubt that nerve-force can be reduced to some form of motion, just as electricity, heat, light and other physical forms of energy may all be shown to be modes of motion. In other words, the so-called "nervous current" which transmits a peripheral impression from the skin or the ear to the central nervous system is no other than an oscillation of the molecules of the axis-cylinders of the nerves. The motion of one molecule is communicated to its neighbour, and so on, until the wave of motion has reached the central organ, just as heat and light waves are transmitted through the air and ether respectively, or electricity through solid bodies. When this motion reaches the cerebral centres, what happens? The same thing is repeated. The motion is taken up by the cells and fibres of the brain. If the peripheral impression has come from the ear or from the eye, the protoplasmic molecules in the temporal lobe in the one case, and in the parieto-occipital lobe in the other, join in the oscillation, and if muscular action ensues, the molecular agitation is transmitted again along the motor nerves to the muscles, where it is converted into muscular energy. Thus far it will be noticed that we have had to deal with modes of motion, purely physical processes, and we have come across nothing like mental activity, nothing like the sounds and visual pictures which are supposed to arise at the instant when the molecular wave reaches the temporal and occipital lobes; and assuredly we never shall see anything of such psychical states as long as we only look at the process from one point of view. Unless we go behind the scenes, as it were, and see what all these molecular processes really are, we shall be as blind to the mental process as Nelson was to the signal for retreat at Copenhagen, when he put his blind eye to the telescope and said he didn't see it.

This failure to find even the first rudiment of mind in tracing the chain of neural motion from the periphery to the centre and back again, is what has led Hughlings-Jackson to fall into the same hole into which Huxley stumbled, when the latter said that consciousness was as completely without the power of modifying the working of the body as the "steam whistle, which accompanies the work of a locomotive engine, is without influence upon its machinery."

So Jackson, with his mind only on the physical process, says that "the visual image, a purely mental state, occurs in parallelism with—*arises during* (not *from*)—the activities of the two highest links of this purely physical chain; so to speak it 'stands outside' of these links." He does not "believe that volitions, ideas, and emotions produce movements or any other physical states. He would not say that a hysterical woman did not do this or that because she lacked will; that an aphasic did not speak because he had lost the memory of words; that a comatose patient did not move because he had lost consciousness. On the contrary, [he] would give or try to find materialistic explanations of physical inabilities."

The logical process by which these conclusions were reached is plain enough, but it reduces the whole matter to an absurdity. All our conscious experience tells us that our feelings are the causes of our actions. This is a fact which each man can prove for himself as often as he wishes to try. The facts of consciousness are as trustworthy as those of the physical world, but all this experience Hughlings-Jackson is willing to pitch overboard simply because, when examining physiological processes by purely physiological methods, he fails to come across anything like a mental state. It is perfectly true that bodily actions are the resultant of physical processes in the brain, but it also may be true that they are the resultant of mental states; and our failure to see this may be due to faulty methods employed in investigating them. At any rate, we are not justified in casting aside either the evidence of our own consciousness or the evidence of physical research without attempting to reconcile the two, however apparently contradictory. This reconciliation can only be accomplished by inquiring more deeply into the *real* nature of cerebral motion.

Cerebral motion is equivalent to molecular oscillation. But what is motion, and what manner of thing are these molecules? A molecule of nervous protoplasm, for example, is something of definite shape and size and colour and density, just like any lump of protoplasm of which, with myriads of other molecules, it is an insignificant part. And all these peculiarities distinguish a protoplasmic molecule from another kind of molecule of muscular substance, for example, which is of different size, shape, colour, &c. But what *are* these properties? Anyone who has given any special attention to this question now-a-days is well aware that all these properties as we know them, are neither more nor less than our own states of consciousness; and that, in fact, our whole knowledge of the material world is nothing more than a series of such states. Further any inferences that we may draw from a study of the material universe are nothing more than inferences that we may draw regarding a series of states of consciousness that we have under certain conditions.

This is a truth that no follower of Herbert Spencer will be prepared to deny, and although it seems to me that few, if any of those who have thought on the subject would dispute the truth of the statement, I am free to admit that daily experience convinces me that the number of even cultivated persons who have thought at all deeply on the matter is exceedingly small.

Our knowledge of a molecule, then, is nothing more nor less than a series of sensations of our own, and when we talk about a material world as being something without kinship with anything else, we are talking nonsense; or speaking more politely, we are making assertions unsupported by the evidence of the most rudimentary experience. For the only objective (material) world we know is a series of conscious states, and therefore something entirely akin to each one's consciousness; while the thing-in-itself we know nothing whatsoever about, and therefore we are not justified in making any assertions about it at all. Beyond our own sensations of grayness, of hardness, of smell, &c., we know no more what a molecule of protoplasm, or any other molecule, *really* is—we know no more what the molecule-in-itself is—than we know whether angels' wings are tied on with pink ribbons or blue ribbons.

It will be well to stop and ponder for a while upon this, to take in its full meaning. Very few of even those who have fully recognised the truth of each individual fact involved have completely realised the full force of all the facts as a whole. Even John Fiske, who never loses an occasion to insist upon this peculiarity of our knowledge of the objective world, and is most intolerant of everyone else who has not grasped it, seems to forget all about it himself when he comes to consider the deductions to which these individual truths lead. Otherwise I cannot understand how he can so entirely misconstrue Clifford as he has done. For John Fiske to eulogise Clifford is to me very much like Robert Ingersoll appealing to the Archbishop of Canterbury to confirm his own heretical opinions.

But can we derive any help regarding the true nature of a molecule from the ultimate analysis to which the physicist seeks to reduce such material bodies? A molecule of any substance, whether gaseous, liquid, or solid, is never at rest, and is always in motion. The direction and extent of this motion differs according as it is a gaseous, liquid, or solid molecule. In a gaseous condition molecules fly about, for the most part, in straight lines; in a liquid, being always in contact, they slide around each other, moving about in all directions through the whole extent of the liquid; while in a solid, although every molecule is in motion, each is confined to a fixed path of limited extent in which it oscillates to and fro, much as a ball suspended from the ceiling by an elastic cord would oscillate up and down. These are the conceptions, at least, which physicists teach us represent the most probable composition of matter. By means of this constant motion of the molecules, many of the properties of a substance may be explained.

It is possible that we may even go further than this. Sir William Thompson has advanced the hypothesis, and supported it by a series of mathematical calculations, that all molecules are but vortex rings (motion) in an incompressible, frictionless fluid—the ether. All matter is then but a form of the ether, and matter differs from the ether only in the size and arangement of the vortex rings. One of the beauties of this hypothesis is that the properties of material bodies, such as hardness, elasticity, &c., are reduced to one common basis, motion, and accounted for by differences in the size and rapidity of rotation of the vortex rings. Without stopping to consider some of the modifications of this theory, which have been suggested to meet the objections that have been raised to it, it is probable that some such conception as this will be the ultimate analysis of matter on the part of physics, and it will be shown that all material molecules are only points of motion in a universal fluid, and that one kind of matter differs from another kind only in the kind of its motion.

Assuming, now, that the results of these investigations are correct, and that atoms and molecules are simply points of motion, are we any nearer a knowledge of the true nature of such bodies? A few moments' reflection will convince any one that we are as far as ever from such knowledge. The problem has been simplified, that is all. Instead of knowing matter as a group of conscious states called colour and hardness, and size and shape, &c., we have reduced our knowledge to terms of one sense—of vision. All the other modes by which we know the object would have been reduced to this one. But our knowledge now is none the less subjective, a mode of our own consciousness. What is there behind these vortex rings? The thing-in-itself? Of this we have no direct knowledge. It is the great unknown universe. What shall we call it? Shall we call it *force?* If we do, we shall use a term which derives all its significance from our own conscious experience, from our states of muscular sense, and tactile sense, and apply it to something of which we have no experience. The idea of force means nothing except so far as it represents certain sensations—feelings of effort and feelings of resistance. And if we use this term to express an unknown element in the universe, we shall endow that element, of which we have no experience and can have no experience, with qualities which have no meaning except as states of mind. Stop for a moment, and try to conceive what manner of thing this unknown universe in which we live is. What is this material world, which we only know as states of mind—as colour, hardness, motion, force, &c.? By what name shall we call it? We cannot call it power, force, activity, motion, for these names all derive their meaning from states of our consciousness, therefore we cannot use them to describe that which is not consciousness, and so far as we know, does not resemble any conscious experience. We have no language to define it because we have no experience of it to give us language, no mental conception corresponds to it. It is the unknown x.

But in spite of this limitation to our powers, there is another way by which we may obtain a hint, if only a hint, of the nature of this object world. But first, let us turn from the contemplation of this material world of molecules to the world of mind. Will a similar analysis resolve states of consciousness into a known and unknown element? If so, we can never understand the relationship between cerebral activity and mental activity. I hold not. We may, as Spencer has endeavoured to do, resolve complex states of mind into simpler states. We may perhaps resolve any state of consciousness into a number of psychical shocks which have blended into a single feeling, just as a musical note may be resolved into a number of individual sounds which in consciousness blend into one. In this way a sort of psychical unit may be obtained, of which all mental states are compounds. In a similar way the material world may be resolved into "simple pulsations, or rhythmical movements of ether-atoms."

But after all has been resolved into psychical shocks, that shock is still a state of mind, and it cannot be reduced by analysis any farther. It cannot be shown to be still further compounded, and more important still, it is not something that, like the physical world, is known in terms of something else. It is known directly and immediately in terms of itself. Therefore it is absolutely known. Mind is an ultimate, a reality—something that we know just as it exists, not a picture of something else. It is known by direct experience. The real nature of the material world we know nothing about. The real nature of mind we know all about. Our sensations of pleasure and pain are really just as we know them.

This being so, my contention and the contention of those who hold the same view is, that the relation between a mental fact and a cerebral fact is satisfactorily explained in the following way:—A mental state of pain is a real fact belonging either to ourselves or to another person. For the purposes of clearness of presentation let us consider another person's pain. This is a real fact. Cerebral molecular motion is a group of conscious states, not in that person but in ourselves, and therefore it is a symbol of a something else in that other person. This something else is his pain. *The pain is the thing-in-itself; the neural vibration is the visual picture which one person would have if his optic* *apparatus could be affected by the mental state of pain in another person.* Mental states are the reality of molecular vibrations in the brain.

To thoroughly realise the meaning of this conception, one must keep vividly before him the fact that no such thing as a "molecular vibration" occurs, as such, in any one's brain. Something occurs there of course. But what we call a molecular vibration is only a group of conscious states which is our mental picture of an apparently unknown activity, and caused in one person by this apparently unknown activity in another. I say "apparently" unknown, for in this case it happens to be known, *i.e.*, it is consciousness.

An important corollary follows from this. It was seen that we know nothing of the real nature of the material "forces" of the world. There is one exception to this, namely, cerebral "force." This we do know. The real nature of cerebral "force" is consciousness.

But if mind is the reality of cerebral processes, it is obvious that the statement with which we started out is true, viz., that there are not two separate processes occurring synchronously side by side, and correlated with one another *in the same individual.* There is only one process—the psychical. The physical is only an objective presentation of it in another person, just as a man pictured in a mirror is only a representation of the real man in the street. There are not two men. The real man is the real agent in the chain of circumstances making up his life. Therefore cerebral processes (which as we know them are a picture) are not transformed into, nor do they precede nor follow the psychical process, which is the real thing.

In a paper like this it is of course out of the question to adduce any extensive proofs of these postulates, even if it were possible to *prove,* in the sense in which this word is ordinarily used in the physical sciences, hypotheses of this kind. I have already set forth, as I believe, in my work on "The Nature of Mind," elaborate and cogent reasons on which this explanation of the relation of the mind to the brain rests. At best all that can be asked is that such an hypothesis shall satisfactorily explain all the conditions of the problem, and that it shall not be incompatible with any known fact. If it shall be found that there is no known fact with which it is not in harmony, and that it does not conflict with any known law, the theory must be accepted as satisfactory. This we, who hold it, contend to be the case.

It makes comprehensible how a stimulation of the body "gives rise to a psychical process;" and conversely, how a psychical process occasions a physical process. It is in harmony with and explains the fact of everyone's conscious experience that his feelings and volitions are the cause of his actions, and are not only a sort of "steam whistle" without power of modifying the workings of his body. It bridges over the chasm between mind and matter, and recognises, while it makes intelligible, their known differences. It gives an interpretation of the universe as a whole.

In order to avoid any misunderstanding, and to anticipate a possible criticism, it is proper, even at the expense of prolonging this already lengthy paper, to make one qualification of the statement that bodily processes give rise to mental processes, and *vice versâ*. This qualification must be considered as largely verbal if the real nature of the processes is kept in mind, but still an adherence to the strict meaning of language requires that it be made.

It has always been a difficulty to conceive how motion in a sensory nerve on reaching the brain could give rise to feeling, and again, how feeling could give rise to motion in outgoing nerves. Strictly speaking, this does not occur. "Motion" in a sensory nerve, as has been already explained, is only somebody's group of conscious states, and therefore a mental picture of unknown activities in a nerve. Indeed, a nerve is only a similar mental picture of the unknown. Now it is the unknown activity which occasions feeling and other states of mind, and it is the unknown activity again pictured as motion in motor nerves which is occasioned in turn by feeling. As what we call motion is only somebody's picture or symbol, naturally it cannot cause your mental state. To insist on this refinement of language is, however, pedantic, except for the purpose of calling attention to the facts.

There is one point which it is desirable should receive perhaps some further explanation—at least those who have not yet quite grasped the full meaning of this theory may think it necessary.

If cerebral motion is only a picture of the real thing, say a musical sound, why is it that the picture appears as *motion*, a so-called objective fact, while the real thing is a musical sound? The answer is not difficult, and those who have followed me thus far must have anticipated it.

Let us suppose that one of us should show a kaleidoscope to some one, who not only had never seen such a thing before, but was totally ignorant of the effect of prisms, mirrors, &c., on light—in fact, was totally ignorant of the most rudimentary knowledge of optics, and of the physical sciences. Such a person might be an inhabitant of some uncivilised island in the Pacific. Suppose such a person, looking through the kaleidoscope, to see a wonderful variegated mosaic of colours. We can easily imagine how his curiosity would be excited by what he saw. He would, probably, at once examine the other end of the tube, but only to find there, in a glass box, a lot of little pieces of coloured glass thrown higgledy-piggledy together, and nothing at all like the beautiful mosaic that he has seen. He will, probably, still ask where is the picture he has seen? and if you point again to the little box of broken glass he will not believe you. You tell him to look once more through the tube, and as he looks you turn the kaleidoscope, and he sees a succession of changing pictures, each a symmetrical pattern of brilliant colours. Again he takes his eye from the tube to examine the farther end, but, as before, he finds only a handful of pieces of broken glass, comparatively dull in colour, tumbling over each other without order or method. You tell him that is what he sees, but, incredulous, he shakes his head and will not believe you. He insists there is something inside the tube which he sees. You continue to expostulate and tell him there is nothing there, but he retorts that there must be two things, not one. He sees an ever-changing panorama of symmetrical and orderly designs, brilliant with transmitted light, and you are only showing a lot of dull glass tumbling about, and without brilliancy. He says there are two processes totally unlike one another. He can find no similarity between the two, and therefore is confident there must be something inside the tube. So you take the apparatus to pieces, and show him there is nothing inside but some mirrors arranged in a peculiar way. If you have amused yourself sufficiently with his curiosity, and if you think him sufficiently intelligent, you will probably explain to him why it is that these pieces of glass appear so differently when viewed through the tube. It is an optical delusion. They can only appear as if arranged symmetrically because they are seen through an apparatus which, arranged according to the laws of optics, compel anything seen through it to appear in a form different from that when seen with the naked eye.

So it is with mind and a cerebral molecule. If we could *optically* experience a feeling as a pain, or a musical sound, in ourselves or another person, as we can ideally, that feeling could only appear to us as it would be modified by our optical apparatus—just as the pieces of glass were modified in the kaleidoscope. If our visual apparatus were affected by a state of consciousness in another person, it would be an absolute necessity that we should see that state of consciousness (pain, sound), not as it really is but as a visual image, viz., a molecular motion. The *reality* of that motion would be the other person's state of consciousness. This would be the thing-in-itself. Hence it follows that cerebral molecules and motion, which are commonly said to be correlated in each person's brain with his psychical processes, are only the modes by which *one* person apprehends *another's* conscious states. Consciousness is not correlated with molecular motion in the same individual, but only with states of consciousness (*i.e.*, the mental picture of molecular motion) in another person.

ON A CASE OF ANÆSTHESIA AND TROPHIC CHANGES CONSEQUENT ON A LESION LIMITED TO THE GYRUS FORNICATUS AND PART OF THE MARGINAL CONVOLUTION; WITH REMARKS ON THE SENSORY AND TROPHIC CENTRES OF THE CORTEX.

BY THOMAS SAVILL, M.D.LOND.

Bearing in mind the difficulties which often present themselves in observing aberrations of sensation, and the scarcity of uncomplicated clinical observations of this class, the following case seems worthy of being placed on record. Not that the sensory phenomena here were as unmixed and as clear as could be desired; but the symptoms are as uncomplicated as one is likely to find in such cases, which occur for the most part in the aged. The case, moreover, looked at in the light of recent laboratory investigations, assumes an importance which it would not otherwise have. The very unusual position of the hæmorrhage; and the existence of arterial thickening without renal change, are features of considerable interest though they do not call for further comment in this place.

The symptoms, so far as our present inquiry is concerned, observed during life were loss of sensation, and subsequently the formation of bedsores and œdema, on one side of the body. These were found, when death occurred two years later, to be associated with a lesion occupying the grey matter, and some of the white fibres beneath, of the gyrus fornicatus and part of the marginal convolution of the opposite hemisphere.

Richard Sharpe, eighty-two years of age, a locksmith, was admitted into the Paddington Infirmary on the first occasion in May, 1888. His family history presented nothing worthy of note; his parents had been healthy and long lived. His previous history also was unimportant. He had passed a healthy abstemious life, free from gout, rheumatism, or syphilis. He was at work and in his usual health, when, on *May* 16*th*, 1888, he had his first "stroke." His wife informed us that he was taken suddenly; that he fell down unconscious and remained so for three days; and that on coming round his left arm and leg were weaker than his right.

He was brought to the Infirmary seven days later (May 23), when I saw him for the first time, and made the following note:—"Patient is conscious, but his mind is rather confused and there is some degree of aphasia.[1] *No apparent loss of power in either arm or leg now.* Both patellar reflexes normal. *Common sensation seems to be absent everywhere on the left side and partially on the right, excepting the area around the right elbow joint.*[2] Tongue protruded straight. Incontinence of both urine and fæces. Back and hips very sore—more on the left side, on which side there are superficial sloughs in places. No signs in heart or lungs. Urine, acid, 1018, faint trace of albumen."

The mental condition rendered the examination somewhat difficult, requiring time and care, but I made the examination, and this note myself, and distinctly recollect that, whereas the absence of response to touching and pricking on the whole of the left side was absolute and beyond doubt, there were places here and there on the right side, besides the right elbow where he could feel. It may not be out of place to remark that testing sensation in very old, and in very young subjects is always a little difficult; in the former, partly owing to the slowness of mental faculty and partly owing to senile changes in the skin. It is not, of course, sufficient to trust to reflex phenomena. Patience and sufficient time are above all things needed.

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[1] "Forgetfulness of Words" would have been a better phrase, owing to the confusion of thought.

[2] Where sensation was perfect.

May 31*st*, 1888 (*one week later*).—" Much improvement, both in mind and body. Sensation has returned everywhere, he has no incontinence now."

June 14*th*.—" Has suffered from headache for the past two days ; was sick yesterday. Gets up daily."

June 21*st*.—"Is still improving. Swallows quite well now. Pupils unequal, right larger than left. (He had dislocation of the left lens from an accident early in life.)" The temperature was 99·8 on admission, but afterwards it was about normal throughout his two months' stay in the Infirmary.

He made a complete recovery, and was able to leave the Infirmary and return to his work two months after admission (July 18th, 1888).

It was upwards of a year before I saw him again, but I remember the case distinctly on account of its unusual features, viz., a combination of (1) Anæsthesia, (2) One-sided bed-sores, (3) Confusion of thought and (4) Double incontinence, without motor paralysis. During the ensuing year he kept in good health, except occasionally, to use his own words, his " senses would go away from him all of a sudden while at work."[1]

In the summer of 1889, these attacks became worse, and he was also subject to transient attacks of giddiness and syncope, in one of which he fell down in the street, and was taken into St. Thomas' Hospital, where he remained for a short time. I have been unable to find any record of his symptoms at that time ; but the patient said that he was only in the hospital a little while, chiefly on account of his " fainting " attacks, and had no other symptoms. But after leaving he found that his memory was failing him, and his

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[1] These momentary, transient confusions of thought are very frequent in the aged, and without entering into a discussion of the proofs, I may perhaps here state my adherence to the view that they are due to disturbance of the cerebral circulation. They occur most frequently in subjects with thickened systemic arterioles, a condition which existed in this case to a very marked degree. The case is further alluded to in this connection in a paper on Senile Cardio-vascular Changes, read before the British Medical Association Meeting, at Bournemouth, July, 1891.

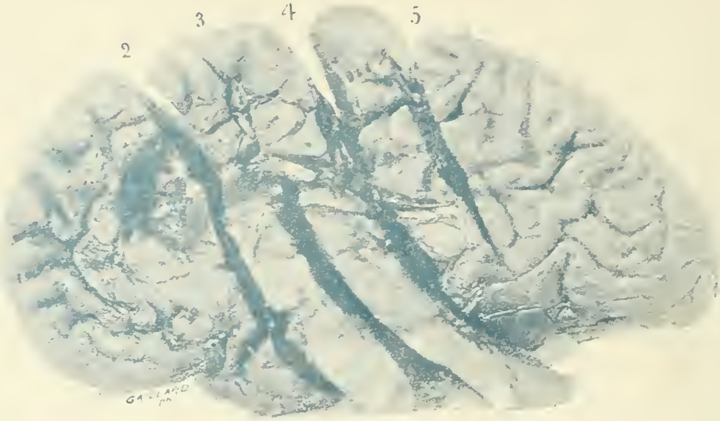

Fig. 1.

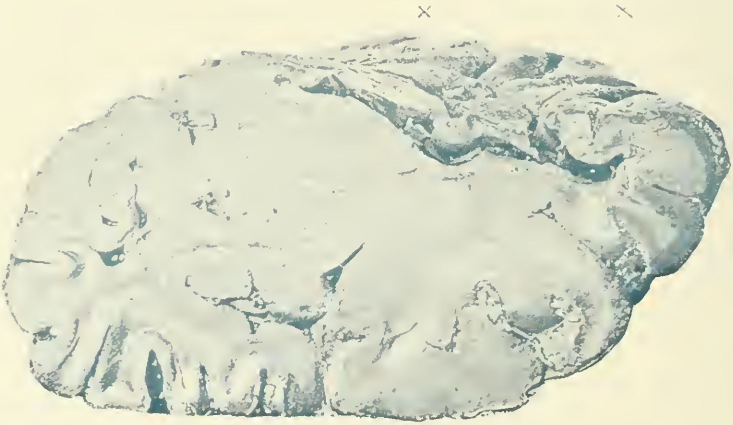

Fig. 2.

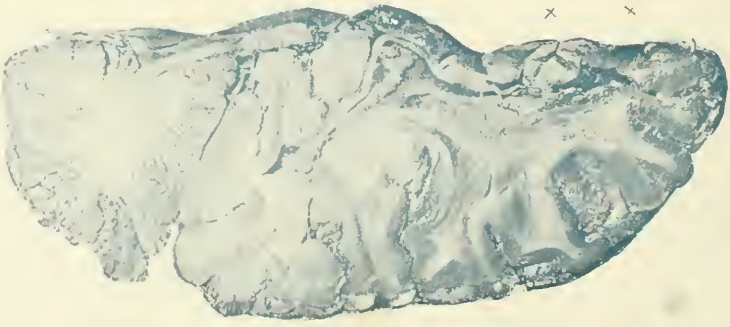

Fig. 3.

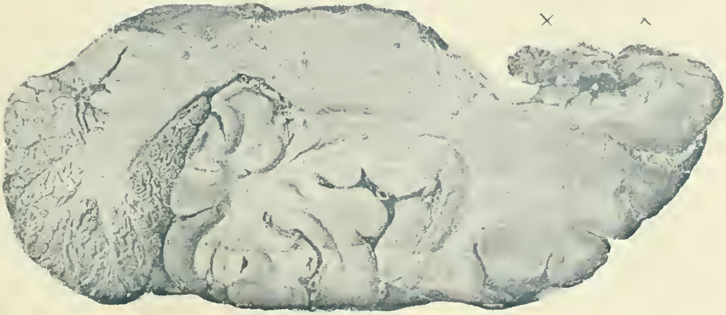

Fig. 4.

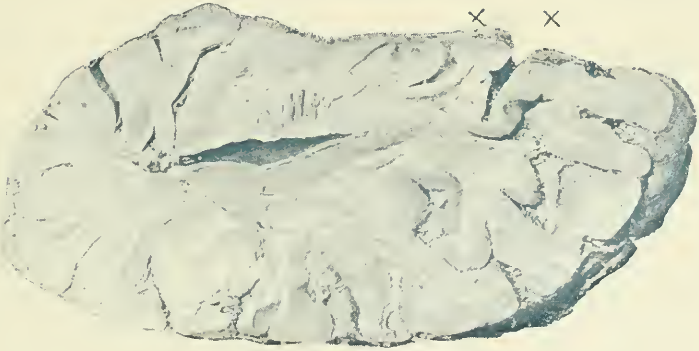

Fig. 5

DESCRIPTION OF PLATES.

Fig. 1.—Right hemisphere of Richard Sharpe. The front end of cavity is propped open by a glass rod. The numbers are opposite the sections from which the subsequent figures are taken.

Figs. 2, 3, 4 and 5.—Vertical transverse sections through the right hemisphere as indicated in Fig. 1, seen from behind; Fig. 2, obliquely across fissure of Rolando; Fig. 3, obliquely across ascending parietal convolution; Fig. 4, obliquely across mid-parietal region; Fig. 5, obliquely across anterior occipital region. Crosses are placed opposite the superior and inferior limits of the cavity.

hands were not so adept at his work as formerly; so he was obliged to seek refuge in the Paddington Workhouse, where he again came under my care, and the case was classed as one of (so-called) arterio-capillary fibrosis, or arteriole hypermyotrophy, as I prefer to designate this condition.

Whilst in the Workhouse, on January 25th, 1890, he was seen to fall. He was not unconscious at that time, and explained that he was too giddy to rise. When examined an hour or so later he was found to be in a semi-conscious condition; he could be roused to answer questions, though not very intelligently, and there was some stiffness of the left arm and leg.

January 29th, 1890 (*four days later*).— The stiffness had passed off, and there was "complete flaccid paralysis of the left arm and leg. Can speak and understand a little now; does not articulate well; is not sufficiently collected to test sensation. Left side of mouth does not move, tongue protruded very much to left. Can shut both eyes. Swallows with difficulty; fluids run out of mouth. Yawns very frequently, and when he yawns, the left (paralysed) arm goes up towards his head. Knee jerks normal on both sides; complete double incontinence; heart and viscera normal; arteries very thick. Temperature last night 99°."

February 28th, (*thirty-fourth day after the attack*).— "Has a slough forming on left heel and left buttock (paralysed side), although he does not lie more on this side, and is most carefully nursed. Right side sound. Left leg becoming contracted."

March 7th (*forty-first day*).—"Has had marked œdema of left hand, leg and foot (paralysed side) for the past week—more of the hand than foot. No œdema of right extremities. Heart sounds healthy but feeble. A few râles at basis of lungs."

March 14th.—"Both legs are becoming more drawn up, and great tendency to bed-sores on bony prominences, chiefly on the left side." The urine was examined from time to time, and always found normal till towards the end of life it became alkaline, ammoniacal, and contained pus. The temperature was normal until the occurrence of cystitis.

In the latter part of April he became drowsy, and wandered at times. The weakness increased, and he died very gradually, apparently of asthenia, without the appearance of any fresh symptoms, on May 1st, 1890, two years after the first seizure, and three months after the last. These are the main points in the clinical history so far as our present inquiry is concerned.

Autopsy, fourteen hours after death.—Serous cavities healthy. *Heart*, 10½ ozs. (after removal of clots). Aortic, mitral, and other valves normal; marked hypertrophy of left ventricular wall (average ⅞ inch. thick); muscular substance firm and under the microscope normal.

Lungs—Emphysematous.

Spleen.—3 oz.; substance rather firm and capsule thickened: substance pale, and the vessels stood out from the cut surface.

Liver—43½ oz.; normal.

Kidneys (right 4 oz., left 5½ oz.) were about the normal size and consistence. The cut surface showed very slight atrophy of cortex; vessels stood out and remained patent. Capsule slightly adherent: microscopically, only very slight increase in the interstital tissue (such as is normal in old age), and the epithelium was healthy, but the arterioles of the kidney, and elsewhere throughout the body, showed a very great increase in the thickness in their walls. This increase was chiefly in the tunica media, slightly in the tunica adventitia also. The increase was due to hypertrophy of the muscular tissue, for by leaving sections in staining fluid for 18-24 hours, the characteristic rod-shaped nuclei could be brought out very distinctly.

The Brain, after being hardened in Müller's fluid for several weeks has been submitted to careful examination. The right hemisphere, when fresh, weighed 24 oz., the left 25½ oz. There was some obvious flattening of the convolutions of the right hemisphere, especially on the superior aspect, along the margin, and in the frontal region. By making a series of vertical transverse sections as shown in the illustrations[1] an elongated, rough walled cavity was discovered, measuring nearly four inches in length, stretching from before backwards, immediately beneath the median aspect of the hemisphere. (*See Plates.*) The cavity is such as follows a hæmorrhage of old date, when the patient survives two or three years. Its walls are very ragged, but not crossed by any trabeculæ or bands. The lesion is strictly localised, and is situated precisely beneath those parts of the cortex corresponding to the gyrus fornicatus and part of the marginal convolution, in their entire extent, and also beneath the anterior half of the quadrate lobule (præcuneus). The cavity stretches from before backwards over the roof of the right lateral ventricle, from which it is separated in places only by the ependyma, without nerve tissue. The cavity is largest at its anterior extremity, where its vertical diameter measures $1\frac{1}{4}$ in., midway 1 in., and at the back it fades away to a point. In the fresh state, it measured nearly four inches in length, antero-posteriorly, and its sides, laterally, were almost in apposition, separated only by a little serum. The median wall of the cavity, formed by what is left of the convolutions just named, is very thin. Thus the grey layer of the Gyrus Fornicatus in its anterior half only measures $\frac{1}{12}$ in. thick, and here there is no white matter at all ; in the posterior half of the gyrus fornicatus grey matter $\frac{1}{7}$ in., white layer $\frac{1}{12}$ in. In the marginal convolution anteriorly, grey matter $\frac{1}{5}$ in., white matter $\frac{1}{10}$ in. ; posteriorly, grey matter $\frac{1}{6}$ in., beneath which is only a faint streak of white fibres. In that part of the quadrate lobule where the cavity is found, the grey matter is $\frac{1}{6}$ in. thick (which is about normal) and white fibres $\frac{1}{10}$ in. thick. *All direct communication between the gyrus fornicatus and the white matter beneath is entirely cut off.* There are no trabeculæ stretching across the cavity. The cavity in front extends to within $\frac{1}{2}$ in. of the vertex of the brain, so that in this position also *all direct communication* between the marginal convolution and the centrum ovale are cut off. The tail of the cavity beneath the quadrate lobule is very small, and not sufficient to sever communication between that structure and the white fibres beneath.

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[1] Executed from photographs which Professor Victor Horsley has kindly had made for the author.

The frontal, central, parietal, and occipital convolutions and the uncinate and hippocampal gyri of this hemisphere, and all the convolutions of the other hemisphere, are quite healthy. The cerebral meninges are somewhat thick. The cerebral arteries are atheromatous, but do not present the hypertrophy of the middle coat found in the systemic arteries. By close inspection of sections through the brain in various directions, numerous minute cavities are to be seen in the white matter of both hemispheres about the size of a hempseed or less. These are apparently the results of miliary softenings. Situated in the right internal capsule is a localised softening of more recent date about the size of a small split pea, obviously the lesion producing the "stroke" three months before death. But this lesion is scarcely large enough to account for all the symptoms present during the last three months of life, which included amongst others transient rigid then complete flaccid left hemiplegia followed by contracture of both lower extremities; and it seems more than probable that inflammatory œdema around the old lesion produced some of the later symptoms, and contributed to a fatal issue. The greater extent of the symptoms on the second occasion would be in keeping with an inflammatory lesion, whose extent would be greater and less well defined than a hæmorrhagic lesion.

It is to be regretted that the spinal cord was not preserved.

Remarks.

There can be no doubt from the foregoing account that the symptoms from which the patient suffered in May, 1888, were due to a hæmorrhage into the right hemisphere just beneath the median surface in the position of the cavity which was discovered after his death two years later. It is a matter for sincere regret that the boundaries of the anæsthesia at that time were not observed with greater exactitude. But those who have tested sensation in the aged will readily appreciate the difficulties encountered, and will share my satisfaction that certain facts were made out. It is, however, quite certain :—

(1) That this patient had completely lost the power of perceiving when the left side of his body was touched or pricked seven days after the hæmorrhage, which cut off all communication between the right gyrus fornicatus, marginal convolution, and anterior part of quadrate lobule with the parts beneath.

The condition of the sensation on the right side is not quite so clear. There was an area on the right arm where sensation was intact, but over the rest of that arm, the right leg and the trunk on that side, the sensation is noted as being imperfect. The tumefaction of the right hemisphere at the time of the hæmorrhage must have been very considerable, and there would undoubtedly be considerable pressure on the hemisphere of the opposite side. Is it possible that this pressure, which would take effect mainly on the corresponding convolutions on the median aspect of the left hemisphere, could give rise to impaired sensation on the right side of the body, if it be conceded that the gyrus fornicatus is the centre for sensation of the opposite side of the body? The anæsthesia after the initial attack passed off in the course of fourteen days.

The mental condition after the second attack did not permit of an investigation of the sensation.

(2) That when he came under observation on the first occasion, seven days after the first stroke, and the anæsthesia was noted, there was no paralysis.

(3) It is also quite certain that although there were abrasions on both buttocks, nevertheless sloughs formed only on the left buttock, none on the right, almost immediately after the initial seizure. The same phenomenon occurred after his second seizure (eighteen months later) when sloughs not only formed on the left buttock, but also on the left heel, and in spite of the most skilful nursing; the right side not being affected until a much later stage of the case.

(4) Soon after the second stroke another, kindred, phenomenon presented itself in the shape of very marked œdema of the left hand and foot, in the absence of heart, renal[1] or

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[1] It will be remembered that the examination of the kidneys after death confirmed this, a fact of great importance.

liver disease; the right side again remaining free. This condition remained till death.

The history of a transient left hemiplegia, when he recovered from three days' "unconsciousness," was obtained from his wife. Although we were unable to verify this ourselves, it is quite likely that it should be so for a small part of the motor area of the cortex, which extends on to the median aspect, must have been involved in the lesion, as well as that part occupying the vertex.

In a case of hemi-anæsthesia with other symptoms, published by Dr. Sharkey,[1] the extent of the lesion, judging by the illustration, was probably deep enough to involve the fibres coming from the grey matter on the median aspect of the hemisphere. Dr. Allen Starr[2] has collected a series of forty-one cases showing cortical lesions with sensory symptoms; and Dr. C. L. Dana[3] has collected 142 cases; which are admirably criticised by Dr. C. K. Mills.[4]

Munk, Luciani, and others hold that the area for common sensation lies in the central region of the cortex, that part beneath the parietal bone.

In some valuable laboratory researches made by Horsley and Schäfer[5] the conclusion arrived at is that the centre for painful and tactile sensations is in the gyrus fornicatus. There is a very striking similitude between many of the experimental lesions made by these observers, and the pathological lesion in the case under consideration. Taken in connection with these experiments the facts of this case become of much greater interest.

Dr. Ferrier[6] says, "I have maintained—and a similar opinion has been expressed by Charcot, Nothnagel, &c.—that there is no necessary connection between cortical lesions of the motor zone and affections of sensibility; and I am further of opinion that the motor and sensory centres are anatomically distinct from each other, though functionally and probably organically connected together. Others (Exner, Luciani, &c.) hold that the sensory and motor centres coincide, and believe that cortical motor lesions affect common sensibility as well as motion." The case under consideration goes a long way to settle this question in favour of the first-mentioned observers.

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[1] *Med. Chir. Trans.*, 1884, p. 265.
[2] *Am. Jour. Med. Sci.*, 1884, vol. 87.
[3] *Am. Jour. of Nerv. and Ment. Dis.*, Oct., 1888.
[4] BRAIN, part 47, p. 385.
[5] *Transactions of the Royal Society*, vol. 179 (1888), B., pp. 1-45.
[6] BRAIN, part 45, p. 38, July, 1889.

But Ferrier in his earlier experiments[1] found that destructive lesions of the cornu ammonis and gyrus hippocampi caused impairment or abolition of tactile sensibility on the opposite side of the body, and located the cortical centres for this form of sensibility in the hippocampal region. In experiments with Professor Yeo in 1884, he confirmed these results, but did not produce permanent anæsthesia.

Professors Horsley and Schäfer remark in conclusion:[2]—"The results of our experiments upon the limbic lobe[3] seem to point to the conclusion that this portion of the cerebral cortex is largely, if not exclusively, concerned in the appreciation of sensations, painful and tactile. This is an extension of the view put forward by Ferrier, who was inclined, as the result of his own experiments, to limit that function to the hippocampal region. Dr. Ferrier, who was good enough to assist at some of our experiments upon this part, has fully accepted the conclusions to which they point.[4] These conclusions appear, however, completely incompatible with the views of most of our predecessors in the field of experimental cerebral research."

The question is undoubtedly beset with considerable difficulty. It is in the belief that every contribution tending to throw light upon the subject, however small, is of value, that I have been induced to publish this case.

Bearing in mind the more permanent character of the trophic and vaso-motor lesions (sloughing and œdema),[5] the case would seem to throw even more light on the centres for these influences than on those for common sensation. I am informed that both Professor Schäfer and Dr. Bradford have observed vaso-motor phenomena to follow experimental excitation of the limbic lobe in monkeys, but I do not know where these results are published. It will be remembered that the trophic symptoms after the first, or apoplectic attack, were only transient, just as the irritative effects of the hæmorrhage were transient; but after the second (inflammatory) lesion the trophic phenomena were both more severe and more permanent, just as the lesion itself was more irritative, more lasting, and fatal.

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[1] Quoted by Dr. Charles K. Mills, BRAIN, part 47, p. 380.

[2] *Loc. cit.*, p. 23.

[3] The limbic lobe in the monkey corresponds morphologically with the gyrus fornicatus and gyrus hippocampi in man (Broca).

[4] "Functions of the Brain," 2nd edition, pp. 341 *et seq.*

[5] The sloughing of the buttock after the *first* "*stroke*" (May, '88), passed off in a week or two, soon after the anæsthesia had cleared up. But the sloughing which occurred on the left buttock and heel shortly after the *second* "*stroke*" remained till death, two months later. The œdema of the left arm and leg came on about March 1st, and remained until death on May 1st.

In conclusion, I submit:

(1) That this case supports the experimental evidence that the gyrus fornicatus, is the centre for common or tactile sensation on the opposite side of the body.

(2) That this loss of sensation, in man, may be quickly recovered from, *e.g.*, in the course of fourteen days, presumably by a process of substitution of centres, the other hemisphere taking on the functions of the parts destroyed; as is known to occur in the motor centres; such substitution in the case of sensation taking place more quickly than in the case of motion.

(3) That a destructive lesion in this position (gyrus fornicatus) may produce loss of sensation without involving loss of voluntary motion, at any rate, to any serious extent.

(4) That a destructive lesion in that position is attended by vaso-motor or trophic changes, of a more permanent character than the sensory changes, in the skin and subcutaneous tissues on the opposite side of the body; and therefore these convolutions may possibly be the centre not only for sensation but also for trophic influences transmitted to the opposite side of the body.

The trophic changes affecting the skin in my case were the sloughs which occurred during the first attack in May, 1888, from which he recovered, and the sloughs and œdema of limbs which occurred after the second attack in January, 1890, from which he did not recover. The latter may have been due either (1) to a permanent interruption of the trophic paths by the apopletic cavity at the time of the first attack, or (2) to the irritation set up by the inflammatory œdema around the cavity at the time of the second seizure.

THE PHRENIC NERVE.

BY JOHN FERGUSON, M.A., M.D. (TOR.), L.R.C.P. (EDIN.).

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THE following short notes may prove of some interest to the readers of BRAIN.

(1) In a case of progressive muscular atrophy, with atrophy of the diaphragm, the phrenics were examined after the death of the patient. There was found a mixed condition of degeneration. Some fibres were completely degenerated, others were undergoing the process of degeneration, while still a third set were quite normal. The inference I deduce from the fact that some of the nerve fibres remained quite healthy, is that the phrenic nerve is not wholly motor. Any fibres of sensory character would not atrophy in this disease.

(2) I operated on a cat, and divided the phrenic nerve on the right side. Three weeks afterwards I opened the abdomen and tested the diaphragm as to its sensation, and found it much reduced on the right side. On examination of the nerve there was found a condition of atrophy involving all its fibres. Here, then, what had escaped in the case of progressive muscular atrophy, had been included in the case of a transverse lesion.

(3) In another cat I operated on the spinal column so as to expose the cord, and the third, fourth, fifth, and sixth spinal nerves. All structures in the way were carefully and thoroughly removed. The posterior roots were exposed from the third to the sixth, and, after being raised by a hook, they were divided just external to the ganglia. The animal was killed on the tenth day after the operation. The examination of the phrenic established beyond a doubt the existence of degeneration in the nerve. A point of note is that only about one-third of the fibres seemed to have undergone degenerative changes. These were, no doubt, the sensory fibres cut off from their ganglia in the experiment. The diaphragm was examined before the animal was killed, and found to be anæsthetic on the side of the operation.

About nine months ago I had a patient who died of an abscess in the liver. There was decided inflammation of the serous membrane covering the diaphragm in contact with the liver. In this case the pain in the back of the neck and out on the shoulder was very intense. This pain was always made worse by movements, coughing, or vomiting. These acts disturbed the diaphragm, and at once started or intensified the pain in the shoulder and neck.

A very excellent and suggestive paper may be found in BRAIN for January, 1888, by Dr. James Ross, of Manchester. At page 353 he speaks, from a clinical point of view, of his opinion that the phrenic is sensory as well as motor. Peter and Henle have both suggested the same thing, but advanced no proofs in support of it.

I think the facts set forth in this paper may be taken as very conclusive proof that the phrenic is a mixed nerve, and that in inflammations involving the diaphragm or the serous membranes attached to it, we may look for sensory disturbances, and often of widely distributed character. The phrenic being a sensory nerve, it becomes apparent at once that irritation of this nerve would be capable of irritating the cervical nerves from which it springs, and thus we would find pain in the back of the neck and on the apex of the shoulder. A similar explanation holds good for the pain in the knee of early hip disease, or in many teeth when only one is unsound.

Clinical Cases.

TRAUMATIC HÆMORRHAGE FROM A VEIN OF THE PIA MATER; COMPRESSION OF BROCA'S CONVOLUTION AND OF THE SENSORI-MOTOR AREA OF THE CORTEX; APHASIA; PARTIAL RIGHT HEMIPLEGIA AND HEMI-ANÆSTHESIA; TREPHINING; REMOVAL OF CLOT; RECOVERY.

BY M. ALLEN STARR, M.D., PH.D.,
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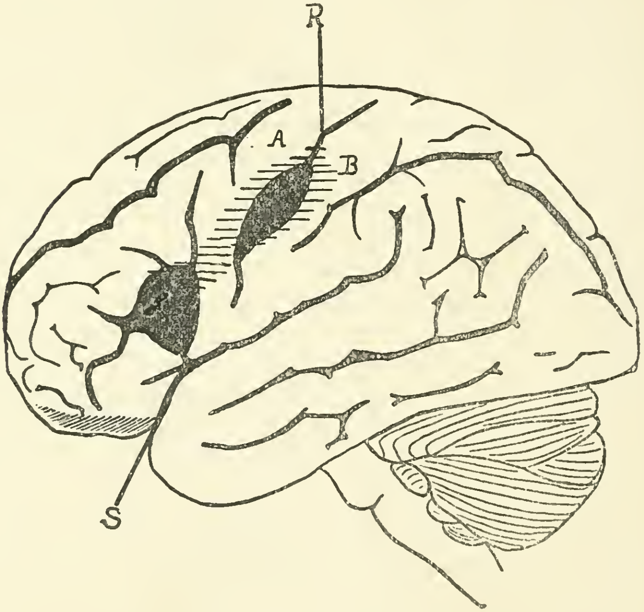
AND
CHARLES McBURNEY, M.D.,
Professor of Surgery, College of Physicians and Surgeons, New York.

History.—A physician, aged forty, was thrown from his carriage on August 17th, 1889. He was slightly stunned by the fall, but had no wound, and was able to help his wife, who appeared to be injured, to reach home. For several hours he seemed to be suffering merely from bruises, and was able during the afternoon to converse with a patient and to administer a hypodermic injection. In the course of the evening, however, he became delirious, then stupid, and for the following three days he lay in a semi-comatose condition. On the morning after the injury he was found to be completely hemiplegic on the right side, and aphasic. When, after a week, his consciousness had fully returned, it appeared that the aphasia was purely motor, as he could understand what was said to him, and could read. The hemiplegia was attended by a partial anæsthesia, the paralysed limbs felt numb, were less sensitive to touch and to pain, but acutely sensitive to cold and to heat. In this condition he remained until December, when he was brought to New York and admitted to Roosevelt Hospital, where he was seen by us.

Examination.—The patient, a large man, was unable to say anything, even "yes" or "no," the vowel sounds being the only sounds produced by effort. He evidently understood questions, and attempted to reply by gestures and by trying to write with his left hand. It was noticed, however, that he was mentally dull, so that the result of tests of muscular sense were not satisfactory, though he could be tested for tactile and pain senses, which were found to be impaired on the paralysed side. He was emotionally unstable, laughing too readily, and at times appearing to be much depressed. Ophthalmoscopical appearances were normal. Sight and hearing were normal. The right hemiplegia was partial. He could turn his eyes in all directions, but could not turn his head to the right. His left pupil was one-third larger than the right pupil, but both reacted normally. His face was slightly flat but not paralysed, and his tongue protruded straight. His arm was almost totally paralysed, the only motion possible being a slight abduction at the shoulder. His leg could be moved a little at the hip and knee, and when held up by two persons he could drag the leg forward a little, but could not stand alone. The hand was flexed and pronated, the leg was extended; both were very rigid; and all the deep reflexes were greatly exaggerated, so that wrist and finger clonus, as well as patella and ankle clonus, were easily produced. He controlled his sphincters perfectly. There were no scars upon the head.

Diagnosis.—It seemed probable that as a result of the fall there had been a rupture of a small vessel, from which very slow hæmorrhage had taken place; the vessel was thought to be a vein rather than an artery, because of the very slow development of the symptoms. The situation of the clot was thought to be upon the surface and not within the left hemisphere, and it was located upon the posterior part of the third frontal convolution and over the anterior central convolution in its middle third, as shown in the diagram. A flat clot in such a situation might fail to compress the face area, and could produce an absolutely total motor aphasia; while a sub-cortical or capsular clot could hardly produce total permanent motor aphasia and paralysis of the arm without producing paralysis of the face and tongue. On the strength of this diagnosis it was thought best to operate.

Operation.—On December 13th, 1889, Professor McBurney trephined the skull. The trephine was applied at a point one inch and seven-eighths behind, and seven-eighths of an inch above, the external angular process of the frontal bone, and the opening was then enlarged by the rongeur forceps upward and backward, the dura being laid bare over an ovale area three by two inches. The dura did not pulsate. On opening the dura, the pia was found to be very œdematous and discoloured, and the surface of the brain was separated from the dura by a space of half-an-inch in depth, and did not pulsate. The clot was seen lying beneath the pia upon the posterior part of the third frontal convolution, and extending over the anterior central convolution in a thin layer (marked in lines on the diagram) into the fissure of Rolando, which was filled with a larger clot lying in the situation shown in the diagram, and extending downward so as to fill up and distend greatly the *cul-de-sac* at the lower end of the fissure. The clot had not covered the lower third of the anterior central convolution, and had not reached the upper



quarter of the fissure of Rolando. The brain, at a distance of an inch about it, appeared to be healthy and pulsated, but the parts of the cortex on which the clot lay were pulseless and stained a yellowish red. After the pia had been incised the clot was removed little by little by fine sponges—at least a drachm of partly organised clot being taken out of the fissure of Rolando. The retraction of the brain from the skull was even more evident when the operation was complete. The operation was done under strict aseptic precautions, and was not followed by any rise of temperature. The wound was dressed in the open method, being packed with gauze, and drainage tubes being inserted. On renewing the dressings three days subsequently to the operation it was found that the entire surface of the brain was pulsating normally, and that the brain surface presented a normal colour and appearance. After a week the drainage tubes were removed, and after three weeks the wound had entirely healed, the level of the scalp at the bottom of the cavity being an inch below the normal level.

Result.—On the evening of the day of operation the patient said yes and no for the first time since the injury—and since that time his recovery of speech and of power has been progressive. After a few days it was noticed that he was much more intelligent, and no longer emotional. Power in his leg began to increase very soon, and two months after the operation he was able to walk with a cane. The return of speech was slow but continuous, and it appeared as if he were learning a new language. He repeated words after another until he had learned them. He talked in monosyllables for the first three months, then he began to put two words together, and then used short sentences of three or four words. At the present time, fifteen months after the operation, he is practising medicine in his native town, is able to walk without a cane, can name at once any object shown him, but in talking uses only a few words at a time, not speaking fluently, writes with his left hand, and were it not for the loss of power in the right hand, might be considered fairly well. The paralysis of the right hand remains, and with it a marked diminution of tactile and pain senses, two points being felt as one when two cm. apart on the tips of the fingers. The muscular and temperature senses are perfect. He can move the arm and forearm in all directions with much force, and can supinate the hand, but movements below the wrist are very slight, the fingers being flexed and rigid. The reflexes are much less exaggerated than before the operation, clonus not being elicited excepting at the ankle; the head can be turned in any direction, and the pupils are equal.

Remarks.—Trephining for aphasia is not uncommon after such injuries as fractures, blows, or stab wounds in the temporal region.[1] There are very few cases reported however in which it has been done when no external injury was present. That it is necessary to distinguish motor from sensory aphasia, and thus determine the place for trephining, and that it would be somewhat venturesome to trephine if the aphasia were only partial, are facts which require no comment. The case reported demonstrates the possibility of successful removal of clots from the cortex, and the necessity of early surgical interference. The persistent paresis in the patient's hand is doubtless due to the fact that the pressure exerted upon the convolutions bordering that part of the fissure in which the clot lay produced permanent nutritive changes in the arm area. The case also indicates the existence of tactile sensory functions in the cortex of the motor area, and thus supports the conclusions reached by one of us in 1884[1] and confirmed by Dana[2] and by Horsley[3] in 1888.

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[1] See Turner, *Lancet*, 1887, No. 3. Singer, *Prager Med. Wochenschr.*, 1887. No. 6. Ball, *Brit. Med. Jour.*, 1888, p. 748. Mouisset, *Lyon Medical* 1889, p. 204.

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[1] Starr, The "Sensory Tract in the Central Nervous System," *Jour. Ment. and Nerv. Dis.*, July, 1884.

[2] Dana, "Transactions Amer. Neur. Assoc.," 1888.

[3] "Transactions Congress of Amer. Phys. & Surgs.," 1888. Discussion on Localization.

RECENT TUBERCLE OF THE PONS VAROLII (WITH POTENTIAL DOUBLE CONJUGATE DEVIATION OF THE EYES); AND OLD CONGESTED TUBERCULAR MASS IN THE CEREBELLUM.

BY J. S. BRISTOWE, M.D., LL.D., F.R.S.

DANIEL MATHEWS, a labourer, forty-nine years old, was admitted into one of my beds on the 12th March, 1890. He stated that he had enjoyed uninterrupted good health, with the exception that in 1881 he was under Mr. Croft's care for some disease of the shoulder, attributed to a fall, when a piece of bone was removed, and he suffered also from some abscesses in the abdominal wall which have left scars. He had been a heavy drinker, but denied having had syphilis.

His present illness began seven weeks before admission. He first complained of a noise in his head as of a bellows blowing, and of numbness in the left thigh and coldness in the left shoulder. Since then the noise (which has been mainly in the left ear) has increased in loudness and become constant; he has suffered from headache, referred mainly to the back of the eyes; he has complained of giddiness and inability to walk straight; he has occasionally seen double; he has lost the power of biting on the right side, and his articulation has become impaired; paralytic weakness of the left side has come on; and he has been troubled with cough.

State on Admission.—He is a fairly healthy looking man, answers questions clearly, seems to be quite sensible, and complains of the symptoms above enumerated. His face presents an unsymmetrical appearance, which at first led to the belief that there was some weakness of the right portio dura, but further examination satisfied us that if there was any such paralysis it was extremely slight. The right masseter and temporal muscles, however, are completely inactive, so that he cannot bite with firmness or masticate on that side, and on opening his mouth widely the lower jaw is thrust over towards the right. There is also marked impairment of sensation over the upper half of the right side of the face, and over the whole of the left half of the face and head and neck. He moves his head freely, but there is conjugate deviation of the eyes to the left. He can with an effort bring them to the middle line, but not beyond, and they always soon resume their original position; there is no nystagmus. The pupils (of which the left is slightly the larger) act both to light and to accommodation; the fundi are normal, and, excepting that he has double vision for all distant objects, his sight seems good; there is no colour-blindness. He complains of incessant loud rumbling noises in both ears, but mainly in the left, and his hearing is undoubtedly impaired, although he hears all that is said in ordinary conversation. The sense of smell is quite normal, but sensation is much blunted not only in the lips, but also through the whole interior of the mouth and the tongue, and he distinguishes flavours with difficulty. Sugar he scarcely recognises, and quinine and tartaric acid he only tastes after some time. The tongue is not paralysed, and the soft palate is symmetrical, but is motionless when he articulates, and displays no reflex movements. His speech is indistinct, but his voice has tone. The left arm and leg are both markedly enfeebled, the leg being the feebler of the two, but they are neither wasted nor rigid. There is almost complete loss of sensation through the left half of the body up to the mesial line. The plantar reflexes and knee jerks are equally brisk on both sides, but no clonus are obtainable. The patient has a slight cough, but there are no physical signs of pulmonary disease, and the heart and the abdominal organs seem all healthy.

During the month following admission his general condition showed no great change, but a few additional facts and some advance or change in his symptoms were observed. He continued to complain of more or less headache and of noises in his ears, which latter, however, subsided to a large extent in the left ear, but became very loud and distressing in the right. Moreover his deafness increased, so that he could hear a watch on the right side only when it was brought into actual contact with the pinna, and on the left side not more than two inches from the ear. The symptoms referrible to the affection of the right fifth nerve remained unchanged, but during the latter part of the month weakness of the right portio dura gradually became developed, so that at the end of this time the right eye could not be closed and the mouth was markedly drawn over towards the left. It was observed, even before the facial paralysis came on, that he kept his right eye habitually open, whereas the left was always either closed or winking. This phenomenon, seemed to be voluntary, and to have for its object the avoidance of the confusion of vision resulting from the simultaneous use of both eyes, the selection of the right for use being probably determined by its insensibility to reflex impressions. The numbness of parts inside the mouth, and impairment of taste remained unchanged, as also did the sluggishness and the absence of reflex action in the soft palate. It was thought, on one or two occasions, that the left palatal arch was more elevated than the other. Also during the latter part of the month the patient experienced a good deal of difficulty both in masticating and in swallowing, food occasionally causing him to choke, and liquids occasionally finding their way into the nose.

From about the middle of April to the middle of May the patient lost flesh and strength, and suffered a good deal from headache referred mainly to the frontal and parietal regions, from choking after food, and from cough; the noises in his ears continued, and all the paralytic conditions which had come on continued without much change. But several additional symptoms accrued. On the 11th April it was observed for the first time that the deviation of his eyes to the left had disappeared, and that he now looked directly forwards. At first it was supposed that this was due simply to the subsidence of his eye trouble. But further examination revealed the fact that, while he could move his eyes quite freely upwards and downwards, he had no power whatever to move either of them in the slightest degree either to the right or to the left of the middle line. It seemed, in fact, that conjugate deviation to the right had become added to the already existing conjugate deviation to the left, and that between the opposing forces the eyes were held in the middle line. Thenceforth this phenomenon persisted without change. On the 27th April it was first distinctly ascertained that he had lost power in his right arm, and a little later the right leg showed signs of weakness. It may be added here that these limbs never became so much affected as their fellows, and that it was never clearly made out that there was right hemi-anæsthesia. On the 6th May his evacuation for the first time passed away involuntarily, and this incontinence continued ever afterwards. On the evening of this day his cough was very violent and incessant, and in the course of it he became almost asphyxiated, and he remained in a condition of extreme danger for some hours. About this time, also, inflammation of the right conjunctiva came on.

From the middle of May to the middle of June he still continued to lose flesh and to become weak; but there was no noticeable change in, or addition to, his paralytic symptoms. His speech, however, became extremely indistinct, partly, no doubt, owing to accumulation of sticky saliva in his mouth; and he became extremely talkative, chatting constantly to himself and making many odd remarks to those about him. It was thought, but it was difficult to be certain, whether or not he had delusions, but he could easily be made to answer questions, and he always answered rationally. During this month he ceased to complain of headache and of noises in his ears, and indeed the hearing in his right ear improved somewhat. The inflammation of the right eye continued, suppuration took place, and the cornea became hazy and ulcerated. But although the inflammation continued for some weeks, and had not wholly subsided by the time of the patient's death, the eye was not destroyed. Towards the end of the month some inflammatory swelling took place in the right parietal region.

During the latter half of June, although the patient still continued to get weaker, his mental condition improved; he became quieter, and he swallowed with less difficulty than before. On the 27th of the month it was noted that he still swallowed without much difficulty, but that he was drowsy and disinclined to talk, and that his speech was almost unintelligible.

After this he gradually sank, he became weaker and weaker from day to day, more and more quiet and disinclined to take notice of what was going on, his replies to questions becoming apparently incoherent, and his breathing grew noisy and difficult. He died on July 4th at 8 a.m.

It may be added to the above account that his pupils always acted to light, the left usually being rather the larger; that no optic neuritis was ever observed; that his temperature (excepting when on two or three occasions it was raised from some inflammatory cause, and on the evening before death, when it rose to 102) was generally normal or sub-normal; and that the immediate cause of death appeared to be accumulation of mucus in the air-passages.

Autopsy.—Unfortunately the post-mortem notes of this case have been lost. The examination was conducted by the late Dr. Gulliver, who forgot to send them to the Registrar, and they have not been found among his effects. I was, however, present at the examination, and the more important facts that were observed have remained impressed on my memory.

The brain generally was healthy, and there was no congestion or excess of fluid, or disease of arteries The pons varolii looked unduly plump, especially in the left half, the surface otherwise being normal. But it was hard and resistent, and one section was found to have imbedded in its substance a cheesy mass about as large as a good sized chestnut. This was sharply defined, and surrounded on every side by a thin layer of nervous substance. It encroached rather more on the left half of the pons than on the right, and involved the anterior part of this body in greater degree than the posterior. It also caused a little protrusion of the floor of the fourth ventricle. Neither the crura cerebri nor the medulla oblongata were invaded by the growth. Another mass about as large as a hazel nut was imbedded in the surface of one of the lobes of the cerebellum (I forget which). This presented a dense fibrous capsule about half a line thick, and its inner part consisted of cheesy matter, becoming dry, cretaceous, and friable.

In the apex of the left lung were some indurated patches and scars, obviously due to former tubercular disease, and both lungs (especially in their lower and back parts) were congested and œdematous.

There was no further evidence of disease.

Remarks.—The case above recorded was naturally regarded with great interest while it was under observation in the hospital. The symptoms at the time of admission (of which the more characteristic and important were left hemiplegia and hemianæsthesia, anæsthesia of the right side of the face and paralysis of the muscles supplied by the fifth nerve on that side, conjugate deviation of the eyes to the left, noises in the ears with incomplete deafness, and difficulty or uncertainty in swallowing associated with some loss of power in the soft palate) pointed clearly to some disease affecting the pons varolii. The progress of the case was slow, but on the whole confirmatory of the diagnosis; for while the phenomena above enumerated underwent little material change, other confirmatory phenomena accrued from time to time, namely (not to give them in the exact order of their occurrence), partial right hemiplegia, paralysis of the right portio dura, and a condition of the motor mechanism of the eyes which can best be explained on the assumption of antagonism between tendencies to conjugate deviation in both directions. The slight improvement observed towards the end of the case, in respect of hearing and swallowing, seemed to imply that although the region of disease was spreading in certain directions, there were parts of its margin in which some degree of improvement was taking place. The other symptoms which the patient presented had little or no specific interest, and some of them were in a sense accidental. I refer to the garrulity and tendency to ramble which he at times manifested, to the incontinence of evacuations, to the inflammation and ulceration of the right eye (the eye on the side on which paralysis of the fifth and seventh nerves existed), to the inflammation that took place in the region of the right parotid, and to the pulmonary symptoms which seemed largely due to the frequent entrance of foreign matter into the air passage during the act of swallowing, and which on one occasion were so nearly fatal.

I was uncertain, during the patient's lifetime, what the nature of his disease was. I hesitated between softening of the pons secondary to thrombosis of the basilar artery or some of its branches, and a tumour. But on the whole, guided by my former experience[1] of disease of the pons, I inclined to the former view. No history of syphilis could be elicited, which was of course unfavourable to the diagnosis of either thrombosis or the presence of a gumma. The post-mortem examination, however, showed that his symptoms were due to a tubercular growth. Interesting facts in the case are: first, that the brain invasion appeared to be secondary to old disease in the lungs; and second, that the mass of tubercle in the cerebellum must have been of long-standing, had probably never caused symptoms, and had become quiescent—showing that tubercular growths in the brain, as well as in other parts of the organism, may become encysted and inert.

Perhaps the most interesting feature in the case was the conjugate deviation of the eyes to the left, followed later by what I have ventured to name, or rather misname, "double conjugate deviation." The patient was always somewhat difficult to deal with, and it was always difficult to make delicate investigations in which we had to be aided by his own voluntary efforts. This was especially the case in examining the movements of his eyes. During the period of conjugate deviation to the left he could generally with an effort bring both eyes to or near the middle line, thus showing that neither the right external nor the left internal rectus was completely paralysed ; and although he had seen, and did at times see double, he had, so far as we could make out, some power of convergence. But after the eyes had assumed the median position (retaining free upward and downward movement), there was never, so far as I was able to discern, any trace of lateral movement in either direction. The latter phenomenon does not appear to me to be adequately explained by, or even to be compatible with, the ingenious hypothesis suggested by Drs. Hughes Bennett and Savill[1] in their paper on "A Case of Permanent Conjugate Deviation of the Eyes and Head," and I do not myself venture to offer any more plausible solution of the riddle. It is unfortunate that the anatomical examination in this case was never completed, but while it is almost certain that the nuclei of origin of both sixth nerves were involved in the disease and destroyed, it is quite certain that there was no direct implication of the third and fourth pairs.

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[1] Bristowe, "Diseases of the Nervous System," p. 310.

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[1] See Brain, July, 1889, p. 102.

A CASE OF CEREBRAL TUMOUR.

BY H. HANDFORD, M.D., M.R.C.P. (LOND.).

Physician to the General Hospital, Nottingham.

Tumour following injury, and causing paralysis of the left arm and leg, the latter just recovering from paralysis from the same injury; different paths for the conduction of heat and cold.

Case.—J. C., æt. thirty-eight, a miner, was admitted into the surgical wards of the General Hospital, Nottingham, on account of loss of power in the left leg, supposed to be due to an injury to the hip by a fall of coal eleven weeks previously. As the paralysis was evidently not caused by any local condition of the leg, but was probably due to some affection of the central nervous system, the patient was transferred to my care on February 4th, 1890. So far as he knew he had been quite well, strong, and robust until the time of the accident. Though generally crushed, the patient was not aware of any injury to the head, and did not lose consciousness. There was no scalp wound. With the assistance of two companions he was able to rise after the accident, and walk some distance. For the first fortnight he was able to get up for a few minutes daily, but required assistance in getting in and out of bed. After that he was able to sit in an arm chair for some hours a day. About three weeks after the accident the leg began to "jump," and a week later a feeling of "pins and needles" gradually spread up the leg to the side of the trunk, and to the left arm. Pain commenced in the axilla, and passed down the inner side of the arm to the fingers. After a few days the pain in the upper part of the arm greatly diminished, and was chiefly localised in the fingers. He said the pain was very severe, and "as if the finger nails were being torn off." He gradually lost power in the arm. In about a week it became quite helpless, and has remained so ever since. At the same time he became subject to rhythmical movements of the head, which used to be moved from side to side. The movements continued for about five minutes, and recurred once or twice a day. The liability to these curious movements of the head gradually diminished, but they were noticed a few times after his admission. The left leg is always everted, abducted, and semiflexed at the hip and knee joints. There appears to be paralysis of the adductors and internal rotators, and some over action of the opposing muscles, as the leg cannot be placed quite in the normal position. There is much loss of power, but not complete paralysis. When he stands on the right leg the left drops nearly into the normal position, but he can bear no weight upon it. There is no pain in it.

Superficial Reflexes.—The plantar and cremasteric reflexes were brisk and equal on the two sides; the abdominal and epigastric reflexes good.

Deep Reflexes.—The knee-jerks were active and equal on both sides. There was an Achilles jerk on the left, but none on the right; no ankle clonus. Triceps jerk on the left, none on the right; thumb jerk equal on both sides; jaw jerk could not be obtained.

Sensation.—Common sensation was diminished in the left leg generally, but he could feel a firm touch. The contrast with the right leg was very marked. In the left arm common sensation was still more diminished: he could not feel the light touch of a feather at all, and firm pressure on the upper arm was undetected when his attention was at all distracted. Sensation on the right side was unimpaired. On the left side of the neck sensation was slightly deficient. The sensation to pain was more diminished than common sensation in the same areas, but was not absent.

Heat and Cold.—The test was carried out with test tubes filled with water at 45° F. and 150° F., and the patient's eyes were covered. In the left leg he was always correct about the cold tube, which seemed to cause discomfort and made him jump. But usually he could not tell whether the hot tube was warm or cold, although it was hotter than he could bear on the right leg. In the arm the same differences were more marked, and in the axilla he persistently called the warm tube cold. In the neck there was also great diminution in the perception of heat. Over the whole of the right side the sensations were normal. When the hot tube was placed in the left axilla the elbow joint was spontaneously flexed, although he was quite unable to make any voluntary movement, except to a very restricted extent at the shoulder joint. Such involuntary movements in paralysed parts are occasionally observed, and resemble the preservation of emotional movements of the face in hemiplegia, and the utterance of emotional words in aphasia.

The left leg seemed shortened, but careful measurements showed that this was not so. Neither was there any evidence of dislocation or fracture. No localised muscular wasting was detected ; no difficulty in swallowing ; no definite facial paralysis, but some slight weakness of the left side. The speech was always thick, but was not apparently altered by the illness. He had complete control over the rectum, and nearly complete over the bladder, but passed water into the bed a few times.

There was no paralysis of the ocular muscles, but the right pupil was contracted, oval and fixed. The left was about the size of a No. 4 catheter, and reacted both to light and accommodation. There was no hemianopia and no optic neuritis at this time. The tongue was protruded straight.

Electrical Reactions.—All the muscles of the left arm contracted moderately to faradaism. In the left thigh the extensors contracted well, but the flexors, adductors, and all the muscles below the knee reacted very poorly when compared with those of the right side.

In about a week the arm had improved so that he could abduct it nearly to the level of the shoulder. He could not pronate the forearm or move the fingers or wrist.

On February 10th his temperature had begun to rise, and he complained of frontal headache with pain passing horizontally round the head, but worse on the right side. He frequently vomited. There was now some anæsthesia and definite muscular weakness on the left side of the face. Taste was lost on both sides and he could not distinguish salt from sugar.

February 14th.—Temperature normal again ; mentally confused ; vomiting continues ; facial paralysis increased ; can move tongue freely to right but not to the left. He answers questions after a long time, but correctly.

February 16th.—Optic neuritis commencing.

February 24th.—Optic neuritis rapidly advanced ; vessels covered and margins of discs quite obscured. He has some difficulty in swallowing solid food. Holds fluids in the mouth for a long time before swallowing.

February 26th.—No paralysis of the right arm, but very distinct enfeeblement of the *right* leg. Cannot turn the head to the left. Left facial paralysis increased. Never speaks except in reply to questions, and then after a long pause. No convulsions, but slight nocturnal delirium.

Died March 4th.

At the inspection fourteen hours after death the skull cup was somewhat sclerosed, the superior longitudinal sinus was empty, and the other sinuses contained recent black clot. The cerebral veins contained fluid blood. There was no excess of arachnoid fluid, but over the excavation to be presently described, in the left temporo-sphenoidal lobe was a layer of lymph, or decolourised blood clot, about $\frac{1}{8}$ of an inch thick, and in area about 3 inches by 2. It was not adherent to the pia mater. The pia mater was everywhere rather adherent and difficult to remove without tearing the brain substance. In the third quarter from before backwards of the middle left temporo-sphenoidal convolution was a sharply punched-out excavation, irregular in shape, about $1\frac{3}{4}$ inches by 1 in area, and $\frac{1}{8}$ of an inch deep, with pale base and yellow margins (? stained with blood pigment). Two similar but smaller erosions were discovered, one in the posterior part of the left second frontal convolution, and the other in the lower part of the left ascending frontal (fig. 1). After the membranes had been removed a dark-coloured tumour was discovered at the vertex occupying the upper end of the right ascending frontal convolution, and the posterior extremity of the superior frontal, bulging slightly, firm to the touch, and about $1\frac{1}{3}$ inches by 1 inch in size (fig. 2). The dura mater was not adherent to it. On the median aspect it occupied the greater part of the paracentral lobule. The anterior portion of the gyrus fornicatus was bulging, but not

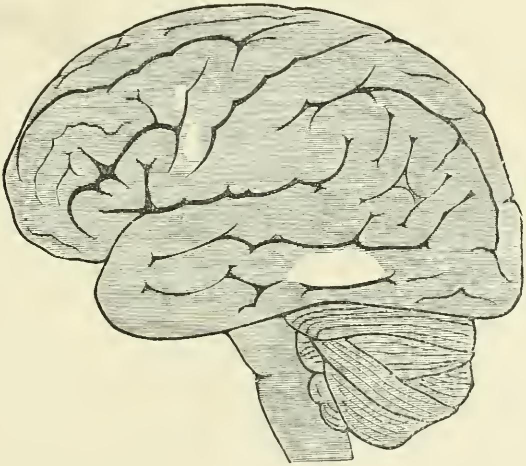

FIG. 1.

The light areas indicate the position of the superficial excavations.

discoloured (fig 3). No new growth was eventually found in this latter place. The right ascending frontal convolution was in its whole length nearly twice as broad as usual, viz., 1¼ inches to 1½ inches. The brain was hardened in Müller's Fluid for subsequent examination. The other viscera were normal. The spinal cord was removed, hardened in Müller's Fluid, and careful examination

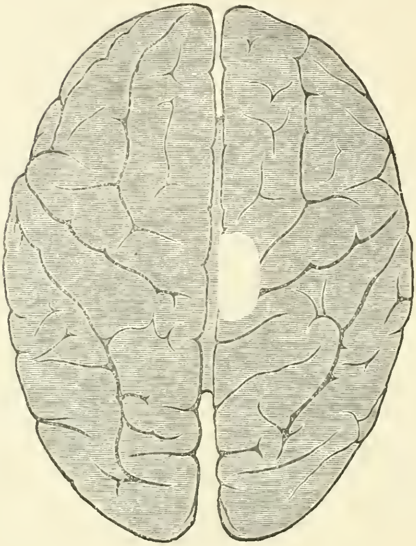

FIG. 2.

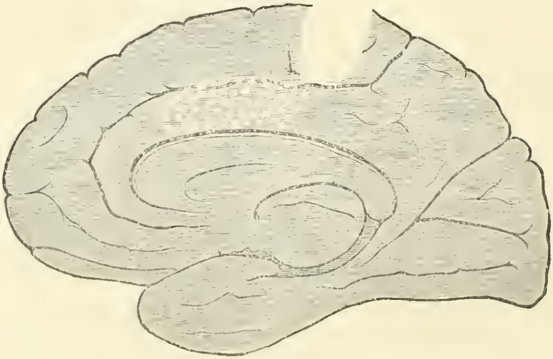

FIG. 3.

The light area indicates the position of the tumour; the dotted area the bulging of the gyrus fornicatus.

revealed no abnormality. There was no dislocation, and there had been no fracture of the left femur.

Eight vertical tranverse sections of the hardened brain were made, as recommended by Professor Hamilton. In the lower part of the right ascending frontal convolution a small area of subcortical gelatinous softening was found. In the fifth, sixth, seventh and eighth sections the tumour was found irregular in outline and occupying the positions marked in the diagrams (figs. 4, 5, 6). There was no trace of a capsule, and no defined limit. The section of the tumour was pale in the centre, and was surrounded by a dark zone which was partly hæmorrhagic. It hardly extended so far as the internal capsule. On microscopical examination of the hardened tumour, it was found to be an angio-sarcoma. There was an abundance of small round cells, some small spindle cells, and a few large round oval nucleated cells. The intercellular substance was scanty and granular and in some places fibrillated. The blood vessels were very numerous, of large size, and with greatly thickened hyaline walls. There were numerous small hæmorrhages, and several areas of degeneration. Many portions seemed infiltrated with threads of fibrine. There were no cells with processes.

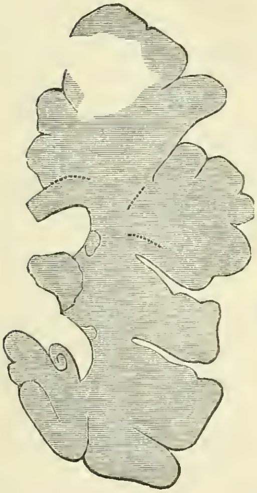

Fig. 4.

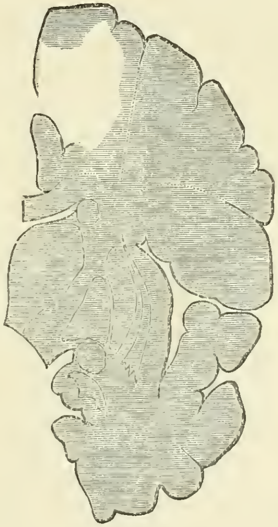

Fig. 5.

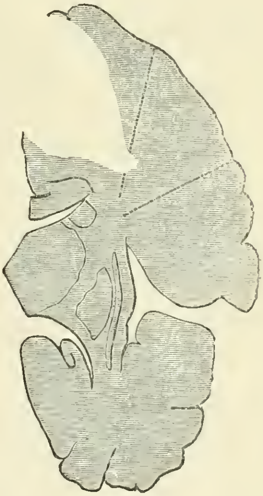

Fig. 6.

The chief point of physiological interest is the support this case gives to the opinion, which now receives a very general acceptance, that there exist different paths for the conduction of the sensations of heat and of cold. In this case the sensibility to heat was greatly impaired, whereas that to cold was, if anything, rendered more acute. Although the tumour did not involve the falciform lobe (gyrus fornicatus) the distinct bulging represented in fig. 3 indicates that pressure effects extended so far. On the mesial aspect the tumour was limited to the paracentral lobule, and did not extend sufficiently far forward to involve, unless indirectly, the area in which the trunk muscles are said to be represented. During life no paralysis of the trunk muscles was noticed, but such a condition is easily overlooked. It is to be noted that the patient usually lay on the right (unparalysed) side, and that at one stage he complained much of pain and numbness in the lower part of the left axilla. The widening of the left ascending frontal convolution was probably of the same nature as the bulging of the gyrus fornicatus, and due to expansion from the pressure of the adjacent tumour. The enfeeblement of the right leg, noticed on February 26th, is not easily explained except on the hypothesis of Broadbent, that parts which are accustomed to act bilaterally are largely represented on both sides of the cerebrum. We must suppose that the right leg was paralysed only so far as it was innervated from the right hemisphere. The superficial cortical lesions in the left ascending frontal and left middle frontal convolutions may have increased the thickness of speech. No effects could be traced to the extensive lesion in the left middle temporo-sphenoidal convolution.

It is difficult to say whether or not this was an instance of a cerebral tumour following and arising in consequence of an injury. If not, the coincidence was very remarkable. The sarcomata are *par excellence* the class of tumours that are apt to arise from injury. Unquestionably the man was quite well so far as he knew, and able to do laborious work up to the date of the accident. He *at once* lost most of the power of the left leg and four to five weeks later further paralysis of undoubted cerebral origin came on. At the end of eighteen weeks death ensued, and a tumour was found. It is not unlikely that the primary loss of power in the left leg was due to a hæmorrhage caused by the accident, and the subsequent paralysis to the growth of the tumour in the injured tissues.

When the patient arrived in the hospital, eleven weeks after the injury, it was for a moment thought possible that the paralysis might be due to neuritis from the injury; but in addition to the presence of the knee-jerk, and the slight alterations in the electrical reactions, the difficulty of explaining the spread of the neuritis upwards through the cord to the upper extremity seemed insuperable. The affection of the arm and face made some form of cerebral disease almost certain, and the development of optic neuritis removed any possible doubt. But I have seen so many cases of severe optic neuritis in cerebral cases, which ended in recovery, that I am unable to accept even an intense neuritis as conclusive of tumour.

In the above case, even with the light thrown upon it after the fatal event, it still remains difficult to correctly apportion the different symptoms to their causes. It was at one time thought that there might be thrombosis of the venous sinuses or cerebral veins, which would have been favoured by the extensive bruising and blood extravasation at the time of the injury. This was found not to be the case, but the erosions on the left side of the brain were evidently of vascular origin, and due either to superficial hæmorrhages, or more probably to sharply localised thrombosis in the superficial vessels. The temporary pyrexia was probably due to the slight meningitis, which was not restricted to the immediate proximity of the tumour. The numerous small hæmorrhages in the tumour did not apparently produce any recognisable symptoms, as the patient presented no sudden variations such as are not uncommon when vascular tumours repeatedly bleed. Although it was felt that the whole of the symptoms were not explicable by the presence of a single tumour, and that the existence of meningitis was almost certain, none the less the great probability that the paralysis was due to tumour was fully accepted. But the total absence of local convulsion rendered it so improbable that the tumour was cortical that no operation was suggested. The extent to which the mesial aspect of the brain was involved showed that removal would have been difficult. The coarse movements at the shoulder and hip joints were very much less affected than the finer movements at the more distant joints. The strange inability to adduct or invert the left thigh was never satisfactorily explained, but there was certainly no physical condition of the joint to prevent it.

The gradual creeping of altered sensations from the axilla down the arm, and the subsequent severe pain in the fingers, both preceding the paralysis, suggest that sensation is represented in the motor area of the cerebral cortex. The absence of hemianopia excluded the likelihood of the lesion being seated in the internal capsule near the "sensory crossway."

NOTES OF A CASE OF DISORDERED SPEECH, EXAGGERATED KNEE-JERKS, &c., FOLLOWING ON SHOCK.[1]

BY H. B. DONKIN, M.B.OXON., F.R.C.P.

The following case is reported owing to its uncommon character:—

J. S., æt. twenty-nine, for the last three years in the Metropolitan Fire Brigade, and formerly a sailor, was in perfectly good health till the beginning of 1890, when he felt much the worry of the telephonic fire-calls. At a fire he suddenly lost his voice, which, however, returned before the fire was over. About this time he used to think he heard the telephone fire-calls when none were sounding. He kept well and quite fit for work until November 13, 1890, when he was at the fire at the Wellington Barracks. He was much excited about the rescue of some children, and in extricating them he fell, through the ceiling, a distance of fourteen feet. He was not visibly injured but was giddy and excited and had totally lost his voice. On admission to Westminster Hospital, two days afterwards, the voice had returned, but he was exceedingly nervous, entirely sleepless, and his limbs were markedly tremulous. There was a slight general want of co-ordination of movement; the knee-jerks, elbow-jerks, and wrist-jerks were excessively marked, and there was distinct, though not great, ankle-clonus on both sides; general sensibility was apparently normal, and there was no affection of the discs or any impairment of the special senses. He improved gradually under the influence of rest, good feeding, and bromide of ammonium, and soon was sent to a convalescent home. He then gradually lost his voice again, becoming soon unable to speak except in a whisper. Suddenly, violent movement of the facial muscles, as of one who stammers badly, accompanied every effort to speak. He returned to his work, but had to leave it again owing to giddiness and neuralgic pain in the head. He was readmitted to hospital on February 6, 1891, speaking alternately in a whisper and a very high-pitched quavering falsetto, and this phonation often took place with the indrawn breath. When he spoke there were marked facial grimaces, and his lips had the appearance of being stuck together, requiring a great effort in separation. With the laryngoscope the action of the cords was seen to be much perverted, abduction taking place with expiration and adduction with inspiration, the movement appearing choreic. He complained of marked *clavus* in the left temporal region. There were several patches of anæsthesia on the legs. At the time of exhibition at the Society the knee-jerks were seen to be much exaggerated, percussion on the patellar tendon or elsewhere causing widespread spasmodic movements of the body generally. The wrist-jerks and elbow-jerks were very brisk, and there was jaw-clonus; ankle-clonus was but very slight. The vocal disorder, as above described, was well marked.

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[1] Shown at the Neurological Society's meeting, March 5, 1891.

This case appears to come strictly in the category of functional disorder after shock. It is somewhat remarkable as occurring in a man of good physique and excellent health, with no family history of insanity or any neurosis, and no previous illness.

Critical Digest.

NEURASTHENIA.

BY DR. PAUL BLOCQ.

(Chef des travaux anatomo-pathologiques à la Salpêtrière).

Translated from the French original by E. F. Trevelyan, M.D., B.Sc.

LIKE hysteria, with which indeed it has many points of relationship, neurasthenia adds to its domain from day to day. Is it that these limits are still undefined, and that a certain number of cases of obscure and doubtful nature are often attributed to it? Or does this increase come rather from the fact that physicians better understand this new disease and recognise its presence in cases where formerly nothing but the appearance, not to say simulation, of disease was believed to exist? Finally, is this neurosis actually more widely distributed as a result of the rapid strides of our civilization? for this advance entails a certain degree of overstrain, and of this, neurasthenia may reasonably be considered the expression.

All these reasons may be appealed to with more or less justice; but whatever be the value of the explanation neurasthenia is none the less tending to occupy a predominant place in neuropathology. This position it owes to the almost unlimited number of its manifestations, to the protean diversity of its appearances, to the complications of its relationships, and finally, to the troublesome tendency recognised in it of "regenerating degenerescence" if such an expression may be allowed: this we shall have to explain later on.

History.—The literature of neurasthenia, however, is relatively poor; not so much because the disease which it represents is, up to the present, wanting in an anatomical substratum proper to it, but because it is destitute of the objective signs which experimental medicine of our times more particularly affects.

In 1869, Beard[1] gave the name of neurasthenia to a nervous state hitherto described under various designations. Among the many names which have been used, we shall mention among others the spinal irritation of Frank,[2] the neurospasmia of Brachet,[3] the general neuralgia of Valleix,[4] the protean neurosis of Cerise,[5] the general hyperæthesia of Monneret,[6] the nervous state of M. Sandras,[7] and the nervosism of M. Bouchut.[8] The new teaching of Beard was not, however, at once accepted; for Krishaber[9] presented his cerebro-cardiac neuropathy as a separate disease, and yet its history clearly corresponds to neurasthenia. In reality it is only from the latest and more complete memoirs of Beard[10] that the real differentiation of neurasthenia from the numerous, more or less similar nervous conditions hitherto confounded with it, and its promotion to the rank of a separate morbid entity, date.

Among the later works exclusively occupied with this morbid condition there is, first of all, a short memoir by Jewell,[11] who scarcely does more than comment on the work of his fellow-countryman. On the contrary, the article entitled *Neurasthenia,* written almost entirely by M. Huchard,[12] in the *Traité des névroses* by Axenfeld, constituted, at least in France the first complete didactic work upon this subject. Weir Mitchell[13] next described a particular form of neurasthenia in women, and he insisted especially on the value of certain physical agents in its treatment. Arndt[1] devoted almost at the same time a very important memoir to neurasthenia. He did not consider it a disease, but "a symptom or group of symptoms representing morbid processes or states, or definite sensations of discomfort," for which he invented a long terminology. But the question presented itself under a totally new aspect in the important publication of M. Glénard.[2] This author described a new morbid condition for which he proposed the name of enteroptosis. He believed that most cases of gastric neurasthenia might be attributed to it. The symptoms of neurasthenia arise, according to this author, from trouble in the abdominal part of the alimentary canal. It is characterised by the prolapse of various organs (liver and kidney), and of the intestines. Particular objective signs are, in such instances, constantly observed. In the treatise of M. Grasset,[3] not less than in the work of Erb,[4] one finds no further mention of the original idea of M. Glénard, of which M. Launois[5] gave a very interesting critical review in the *Revue de Médecine*.

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[1] BEARD. *Boston Med. and Surg.*, 30 Avril, 1869.

[2] FRANK. *De Nevralgia et Neuritide. Univers. praxeos medic. præcipit.* Leips., 1821.

[3] BRACHET. *Recherches sur la nature et le siège de l'hystérie et de l'hypochondrie et sur l'analogie et la différence de ces maladies*, Paris, 1832.

[4] VALLEIX. *Traité des névralgies et affections des nerfs*, Paris, 1841.

[5] CERISE. *Des fonctions et des maladies nerveuses*, Paris, 1842.

[6] MONNERET. *Traité de pathologie générale*, Paris, 1857, t. I., p. 420.

[7] SANDRAS. *Traité pratique des maladies nerveuses*, Paris, 1860, t. I.

[8] BOUCHUT. *De l'état nerveux aigu et chronique ou nervosisme*, Paris, 1860.

[9] KRISHABER. *De la névropathie cérébro-cardiaque*, Paris, 1873.

[10] BEARD. *Neurasthenia (nervous exhaustion). Its nature, symptoms, and treatment*, New York, 1880.—Its causes and consequences, *American Nervousness*, New York, 1881.

[11] JEWELL. The varieties and cause of Neurasthenia, *The Jour. on nerv. and ment. dis.*, Chichago, janvier, 1880.

[12] AXENFELD et HUCHARD. *Traité des névroses*, Paris, 1883.

[13] WEIR MITCHELL. *Du traitement de la neurasthénie*, trad., franc., Paris, 1881.

We shall next mention without further comment a publication by Giovanni.[6]

M. Glénard[7] returned to the same subject confirming his former ideas in a communication to the *Société des hôpitaux,* which provided to M. Féréol[8] the opportunity of making a very remarkable communication. This observer does not pronounce categorically upon the reality of enteroptosis, in the sense of a special disease; but insists upon it as a clinical fact deserving attention, as it may be the source of therapeutical indications. The same year Ziemssen[9] published an article of some importance on neurasthenia.

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[1] Arndt. *Die Neurasthenie (nervenschwæche) ihre Wesen, ihre Bedeutung und Behandlung*, Leipzig, 1885.

[2] Glénard. *Application de la méthode naturelle à l'analyse de la dyspepsie nerveuse. Détermination d'une espèce. De l'entéroptose.* Lyon, 1885.

[3] Grasset. *Traité pratique des maladies du système nerveux*, Montpellier, 1886.

[4] Erb. *Ziemssen Handbuch*, t. XII., art. Neurasthénie.

[5] Launois. *Revue de médecine*, 1887, t. VII. p. 64.

[6] Giovanni. *Sulla neurasthenia*, Cremona, 1885.

[7] Glénard. Société médicale des hôpitaux, 15 mai, 1886.

[8] Féréol. *Bulletins it Mémoires de la Société médicale des hôpitaux*, 5 *janvier*, 1887, pp. 499, 509.

[9] Ziemssen. *Die Neurasthenie and ihre Behandlung*, Leipzig, 1887.

Later, M. Glénard[1] put forward some new considerations upon the diagnosis of enteroptosis.

We ought also to refer to the excellent thesis of M. Lafosse[2] upon *Neurasthenic Headache*, as well as, to a review by M. Lemoinne,[3] and to a work by Glatz[4] on *Neurasthenic Dyspepsia*.

The most important publications of the last years are unquestionably the *Leçons du Mardi*, by Professor Charcot,[5] and the monograph of M. Bouveret.[6] M. Charcot has had on several occasions, both in the first and second years of these lectures, the opportunity of expounding his ideas on neurasthenia. The professor specially considers the frequent combination of neurasthenia with hysteria, and the exclusive part these two affections play in the constitution of the so-called traumatic neurosis.

These views have quite recently been developed with much skill by M. Pitres.[7] The volume just published by M. Bouveret is an excellent and comprehensive work upon the question, and we shall frequently borrow from it. M. Levillain[8] has also produced an important monograph upon neurasthenia, but his work hardly represents anything more than a *resumè* of previous publications, for he adds but very few original remarks to what others have said. The long notice written by M. Vigouroux upon the treatment of neurasthenia by static electricity ought, at any rate, to be referred to here.

Finally, we shall allude to a short monograph upon *Psychical Neurasthenia* by M. Régis.[9] Under this title, (and thus assigning them to neurasthenia), the author describes most of those emotional "obsessions" *(obsessions émotives)* studied particularly by Charcot and Magnan, and attributed by them to hereditary degeneracy, of which these symptoms are the incidental manifestations.

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[1] GLÉNARD. *A propos d'un cas de neurasthénie gastrique*, Paris, 1886.

[2] LAFOSSE. *La céphalée neurasthénique*, Thèse de Paris, 1887.

[3] LEMOINNE. Pathogénie et traitement de la neurasthénie, *Annales médico-psychologiques*, septembre, 1888.

[4] GLATZ. *Des dyspepsies ... et plus particuliérement de la dyspepsie neurasthénique*, Genéve, 1888.

[5] CHARCOT. *Leçons du mardi*, t. I., 1888-1889; t. II., 1888-1889. Voir plus spécialement: 2e, 12e et 13e leçons, 1888-1889.

[6] BOUVERET. *La neurasthénie (èpuisement nerveux)*, Paris, 1890.

[7] PITRES. De la neurasthénie et de l'hystero-neurasthénie traumatique, *Progrés médicale*, 6 decembre, 1889, nº 49, p. 448.

[8] LEVILLAIN. *De la Neurasthénie*, Paris, 1891.

[9] RÉGIS. Les Neurasthénies psychiques. *Journal de méd., &c.*, Bordeaux, 1891, Nos. 36, 37 et 38.

These mental symptoms would have, it is true, according to this doctrine close relations with degeneracy, but only indirectly through their connection with neurasthenia when this latter is degenerative in character. This conception of M. Regis is not supported by any argument of a psychological or clinical order, and appears to us to throw confusion into the question of neurasthenia rather than to enlighten it. At any rate it is a fact that many such "obsessed" individuals present no sign of neurasthenia, and inversely that the great majority of neurasthenics do not suffer from such "obsessions." Finally, the symptoms in question are found associated with degenerative neuropathies such as hysteria, tabes, epilepsy, &c., upon which it does not seem to us any more legitimate to make them depend. Association then, is the only reason which M. Régis has invoked in aid of his theory. M. Grasset,[1] has just traced in the *Montpellier Medical* the history of many cases of neurasthenia, appending some interesting considerations.

Lastly, we may mention that we ourselves have quite recently differentiated, under the name of topoalgia,[2] a certain kind of neurasthenia which is monosymptomatic in character, and which we shall return to in the course of this review.

Ætiology.—Neurasthenia is a widely distributed affection. A rough calculation may be made from the fact that patients suffering from it represent a twelfth part of those presenting themselves at the out-patient department of the Salpetrière.[3] If one considers moreover, that this neurosis is, as a rule, more common among the well-to-do classes than among hospital patients, an approximate idea may be formed as to the average proportion of cases of neurasthenia. Although this disease, especially in its serious forms, is most often of hereditary origin, it is admitted that in certain cases it may develop in a soil hitherto intact from all nervous taint. When heredity constitutes the predisposition, it is rarely the heredity of the same affection, but most often it is the heredity of transformation; and there may be found among its antecedents, insanity, tabes, hysteria, epilepsy.

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[1] GRASSET. Quelques cas d'hysterie male et de neurasthénie. *Montpellier Medical.*—Mai et juin, 1891. Nos. 9, 10, 11, 12.

[2] PAUL BLOCQ. Sur un syndrôme caracterisé par de la "topoalgie" (Neurasthénie Monosymptomatique, forme douloureuse)—Gazette hebdomadaire de médecine et de chirurgie.—Mai et juin, 1891, No. 22, p. 287 et No. 23, p. 268.

[3] According to an abstract made from the lists of diseases diagnosed during my year of office, 1887-1888.

Cases of neurasthenia have been recorded at the early age of 11 to 20, but this is very exceptional. The disease generally makes its appearance between the ages of 25 and 55, the maximum being between 35 and 45. Men are more often affected than women. If the female sex, owing to uterine disorders, offers greater opportunity for the development of this neurosis, men, on the other hand, are more exposed to injury and over-pressure. Certain occupations predispose to neurasthenia, because they demand sustained and considerable intellectual efforts, or because they entail excessive and permanent emotional activity. Thus doctors, whose occupation combines these two troublesome conditions, speculators, engineers, literary men, contribute a striking contingent to the neurasthenical class. Very likely it is because the Americans find themselves in this situation rather than owing to any meteorological conditions that they are affected in relatively larger proportions. The Jewish and Slav races seem to be predisposed to this malady, as they are in fact to most of the neuropathies, without any known reason being assigned for this peculiarity. Gout and rheumatism form a soil most favourable to the development of neurasthenia. In most cases, says M. Huchard, neurasthenia is an "arthritic" neurosis.

The determining causes of neurasthenia are of more importance than in the case of other neuropathies, for the reason that hereditary predisposition may here be absent. They all realise this one condition, cerebral over-pressure, be it brought about directly by excessive activity of the organ, or indirectly in consequence of a debility affecting the individual generally and the nervous system in particular.

In the latter case we may assume a predisposition in the form of a "locus minoris resistantiæ."

Let us now examine the various causes determining the overstrain we have alluded to as the chief cause of neurasthenia. Both fundamental modes of reaction of the nervous system (viz. sensory and motor) may have been overworked. In the first instance, all the passions or excessive feelings may call forth neurasthenia; whether it be love, ambition, filial affection, cupidity, any impediment to their development may set up a consecutive neurasthenia. In the intellectual order, all excess of work may have the same result. The combination of these two sets of causes, intellectual and emotional, will bring about the disease even more easily. This happens particularly in the preparation for competitive examinations.

Traumatism, the preponderating pathogenic influence of which M. Charcot has shown, may in our opinion be compared to a kind of overstrain, its extreme intensity compensating for the shortness of its duration, hence hardly any but severe injuries, and especially those accompanied by shock or violent emotion, are ever followed by neurasthenia. The extreme worldliness of life in large cities, the abuse of pleasures, certain intoxications (as by morphia, ether, cocaine) act in like manner. Among the causes operating through the agency of the general enfeeblement of the individual which they bring about, the majority of the infectious diseases, such as enteric fever, influenza, must first be referred to, and then disorders of the chief systems, and in particular the digestive, genito-urinary, and nervous. As regards gastric disturbance, it seems incontestable that the affection of the general nutrition observed as a result of dilatation of the stomach or the auto-intoxication arising from it, can produce neurasthenia. As to enteroptosis, we shall have to enquire later whether it does not bring with it analogous secondary consequences (a diminution in the lumen of the alimentary canal, and consecutive nutritive disturbances).

Sexual excesses, masturbation, diseases of the sexual organs are also frequent causes of neurasthenia. As to nervous diseases it is difficult to know in the cases in which they precede the appearance of neurasthenia, whether they have played the *rôle* of exciting cause as happens in hysteria developed under the influences of neuropathies, or whether they exist only in the condition of simple morbid associations. However this may be, it is especially in the course of tabes, hysteria, and cerebral syphilis that neurasthenia appears.

Clinical Study.—On account of the many signs, varied forms, and numerous morbid associations of neurasthenia, we have thought it necessary, for the sake of greater clearness of exposition to approach the clinical description in the following order, proceeding to some extent from the simple to the complex. We shall first study isolated symptoms, then the forms produced by the grouping of these symptoms, and finally the association of these forms themselves with other diseased conditions.

(1.) *Signs.*—Among the aggregate of symptoms of every kind observed in the neurasthenical, there are some which are distinguished not only by their constancy, but also by their character, so as to deserve (in analogy to the equivalent symptoms of hysteria) the name of stigmata, using this word in the well-known sense given to it by M. Charcot in his teaching. We shall explain these stigmata first of all, for they constitute under these conditions diagnostic points, and afterwards we shall deal with the variable symptoms.

(*a.*) *Neurasthenic Stigmata.* — These may be arranged thus: headache, rachialgia, intellectual depression, enfeeblement of the bodily powers, and gastric troubles.

M. Lafosse, in the above mentioned work, has met with headache in 44 out of 45 cases. Thus we have to do with a symptom of great frequency. The headache rarely consists, and this is a point of real importance, in true pain. It is rather a sensation of weight and constriction seated in the frontal and occipital regions and comparable to that produced by a heavy, tightly fitting, helmet (neurasthenic "helmet" of M. Charcot). Its habitual localisation is represented by a circular zone starting from the nucha and passing round the temples and forehead, the maximum of discomfort being felt in the nucha. Occasionally the headache only occupies the occiput (occipital patch). It may, on the other hand, be localised between the eyebrows. Finally, it is sometimes unilateral.

The headache occurs most usually in the day. It rarely disturbs the sleep. It disappears during the night, when insomnia is present. It appears at the waking hour, and persists. It is better for a short time after meals, but it soon returns during digestion with greater insensity. The headache is increased by sensorial stimuli—by noise, strong odours, emotion, mental work. When not continuously present as it usually is, these causes habitually provoke it. At times it is accompanied by hyperæsthesia of the hairy scalp, but this hyperæsthesia is rarely to be compared in intensity with that met with in hysteria. M. Huchard has specially insisted upon this symptom. There often exists noises in the ears, heaviness of the eyelids, dimness of vision and even vertigo. Besides this feeling of weight, which is the most constant form, some patients complain at the same time of feelings of emptiness or tension in the head, and occasionally of a sensation as if a foreign body were there following the movements of the head. Rachialgia is less frequently met with than headache, but it may be associated with it. By the fixity of its characters, however, it deserves to be placed among the stigmata of neurasthenia. M. Charcot employs for this rachialgia a name equally striking as that of "helmet" for the neurasthenical headache. He habitually uses the term "sacral patch," and this points out well the usual site and limits of the pain. "The sacral patch is to some degree the counterpart of the occipital or cerebellar patch." (Charcot.)

The dorsal pain consists of a sensation of pressure or heat rarely amounting to true pain. Sometimes there is, however, sharp pain resembling that of neuralgia. We have habitually noticed that these painful phenomena are accompanied by some objective troubles of sensation, anæsthesia or hyperæsthesia of the skin. In the latter case the patient hardly tolerates the least touch, even the rubbing of the clothes. At other times deep pressure upon the spinous processes is painful. The site of predilection of the pain is n the sacral region, but it may be localised in the cervical or lumbar region, or it may even occupy the coccyx. Rachialgia is found more frequently in woman than in man. As to its duration, it may be either temporary or permanent.

The chief troubles by which the mental depression of the neurasthenical manifests itself are diminished power of attention and weakening of the will. Intellectual work is generally trying, and application to any subject difficult. There is often a mental inertia, a kind of cerebral fatigue contrasting strangely with the conversational animation. Diminution of memory is habitual, consisting in a retrograde amnesia affecting proper names especially. The alteration in the power of attention makes reading difficult. The enfeeblement of the will is especially marked in cases of traumatic hystero-neurasthenia. The patients are unable to work intellectually or manually, and they experience a marked sadness and discouragement on that account. The character is changed, so that the patient is dull, dejected and sulky. Response to emotion is excessive. A mere trifle affects him. He shuns society, and seeks isolation and solitude. These mental troubles, taken as a whole, are constant, but they undergo great variations in intensity according to the individual cases.

The diminished strength of the patient must be mentioned as being among the most common signs of neurasthenia. Under these circumstances it does not only consist in a sensation of prostration and lassitude, but there is a real weakness which can be demonstrated by the dynamometer. Without there being a trace of paralysis present, and when the patient is able to execute without apparent difficulty the various movements asked of him, the dynamometer shows a difference of twenty to sixty degrees from the normal.

Sensations of fatigue, and depression in neurasthenia are felt particularly in the early morning, after emotion or moderate physical exercise. It may lead the patient to pass the greater part of his time in lying down. Gastric disturbance is rarely absent from the symptoms of neurasthenia. M. Bouveret recognises two kinds or rather degrees of it, according as to whether it is accompanied or not by objective signs of the enteroptosis of M. Glénard. In the first case the appetite is generally diminished, the tongue a little coated, and the ingestion of food is quickly followed by distension of the stomach, leading the patient to loosen the clothes.

The distension extends from the epigastrium to the rest of the abdomen, and is accompanied by sensations of malaise and fulness. Abundant eructations supervene, and at the same time flushes of heat mount to the face. This trouble lasts for two or three hours or even longer. Constipation is habitual, and accompanied occasionally by phenomena of auto-intoxication, or more frequently by colitis with glairy stools. From this it necessarily follows that the patient presents an earthy tint in his complexion, and such wasting as may suggest cancer of the stomach or rectum. We have ourselves had the opportunity of seeing both one and the other mistake made by distinguished practitioners. By methodical examination one finds in the severe forms of this gastric disturbance, a flaccidity of the abdominal walls, and the various signs of enteroptosis to be referred to later.

(*b.*) *Variable Symptoms.*—Neurasthenia is remarkable for the great number of its manifestations (non morbus sed morborum cohors) for the multiplicity of suffering, cramps, visceral pains, nameless discomforts, so as to constitute a condition described by a physician well-known for his neurotic disposition, as " the punishment of the nerves " (supplicium neuricum). With these patients the state of suffering is a general one. All their organs may be affected one after the other, and yet none of them undergo any coarse change. It results from this that there is often no possibility of localising the mischief, either on the part of the patient or even the physician, and that one may say of neurasthenia what Mead said of hypochondriasis, " non unam sedem habet sed morbus totius corporis est." These words admirably depict the difficulties almost always encountered in the examination of these patients; and which we meet in endeavouring to give an account of the symptoms. We shall describe these under each organ, at the same time pointing out their clinical importance.

Cerebral symptoms. We have already mentioned some disorders which by their frequency merit the name of "stigmata." Disturbed sleep is also one of the most common. Rarely is sleep more prolonged than it should be, for one cannot look upon the somnolence which often torments the neurasthenical after food, as true sleep. Most commonly we observe obstinate insomnia, which at times resists remedies most effectual in other cases. Vertigo is a frequent symptom of neurasthenia. It comes on in paroxysms during fasting, and diminishes after food, or again it may be almost continuous giving rise to slight staggering.

It is not exceptional for the neurasthenical to present certain psychical phenomena which are known under the name of "incidental symptoms of degeneracy," agoraphobia, claustrophobia, pathophobia, &c. We have seen that M. Régis would have it that these perverted emotions are veritable symptoms of neurasthenia, and that he went so far as to group them together under the name of psychical neurasthenia ; but we think the conception is not justified, and in our view these fixed ideas only belong to neurasthenia in the sense of complications. They do not result directly from the illness, but they appear in its course, complicating the clinical picture in the same way as they are observed in the tabetic or hysterical. In all these cases they are engendered from the common source of hereditary neuropathy of which they are characteristic.

Yet we cannot say the same of the hypochondriacal ideas which are almost constant among these patients. The neurasthenical, says M. Pitres,[1] is a restless reasoner or writer, and at bottom a noso-maniac. He is occupied with the symptoms he experiences beyond all reason. He believes himself affected by some organic and incurable disease. He feels his pulse, palpates himself, examines and studies himself. He delights to talk about his illness, and to recount his sufferings. Some very significant peculiarities must be added. These patients have mostly consulted, and read all the books on medicine they have been able to find. On the other hand they have the custom of writing down their sensations in long memoranda which they hasten to read, and to explain to the physicians whom they successively consult. This last detail is characteristic in that it almost suffices to establish the diagnosis.

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[1] Pitres, loc. cit.

Objective troubles of sensation are according to our opinion not so rare as some authors make out. For these hardly anything but subjective symptoms exist. We have proved in nearly all these cases a more or less regular and localised alteration in cutaneous sensation at the site of the pain, consisting in a loss or an increase, whenever we have looked for it. Not infrequently all the integument may be the seat of a true hyperæsthesia. *Subjective* troubles of sensation are innumerable, and assume very different forms both in their character and situation. Painful crackling in the vertebral column, neuralgic, or more or less lightning pains, numbness, and sensations of heat and cold.

The organs of special sense are frequently affected. As regards sight they may consist in heaviness of the eyelids, weakness of accommodation making reading impossible. In hearing there may be hyperacousia, buzzing and whistling in the ears. Taste and smell may present remarkably increased susceptibilities. Thus certain odours may produce at times attacks of headache and vertigo.

Besides the general *motor* weakness, there remains to mention the frequency of cramps and muscular tremors in the lower extremities at the time of going to sleep, and finally the phenomenon known under the name of giving way of the legs.

Circulatory disturbances are illustrated by the neurasthenic angina pectoris described by Landouzy.[1] M. Huchard[2] has differentiated it from true angina pectoris, basing the distinction on the absence of effort as an occasional cause, on the return, sometimes periodically, of the attack, and its longer duration on the more marked agitation of the patient and the lesser intensity of the agonising sensations. There may also be observed in the neurasthenic attacks of palpitation and of frequency of the pulse, which latter is at the same time unstable according to M. Huchard's expression.

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[1] Landouzy, *Progrès Medical*, September, 1883.

[2] Huchard, *Maladies du cœur et des Vaisseaux*, Paris, 1889.

Vasomotor troubles are, so to speak, constant. Among most patients, the meningeal *tache* is readily obtained, but, moreover, vascular spasm leads to coldness and pallor of the extremities. Owing to its intensity the sensations of cold referred to above may be attributed to it.

The respiratory system is less often involved. Attacks of dyspnœa are rarely observed, simulating asthma. A peculiar character in the voice, by which it becomes feeble, confused, and drawling, should be mentioned (Beard).

The generative organs also present functional disorders, consisting in seminal losses and impotence. The bladder is most often unaffected, but a true oxaluria has been described and an excess of urates and uric acid in the urine. This latter, if at all constant, would acquire a great diagnostic importance.

The various secretions are at times more or less affected, watering of the eyes, salivation, profuse sweating of the extremities, and on the other hand dryness of the skin and mucous membranes have been described. When treating of the stigmata, we pointed out the principal signs observed in the digestive symptoms. We shall not return to them here, for we purpose completing what has already been said about them in the following paragraphs on the varieties of the disease.

(2) *Varieties.*—Many forms of neurasthenia have been described owing to the fact that the clinical appearances are so varied. This diversity results not only from the grouping of the symptoms just described, but owing to the preponderance of some of them.

Passing by the inexhaustible nomenclature of Arndt, we may mention that M. Bouveret describes as types a feminine, a traumatic, and a general neurasthenia ; and that he distinguishes, moreover, a cerebral form or cerebrasthenia, a spinal form or myelasthenia and an acute form. On the other hand Pitres recognises the following kinds :—(1) Cerebral ; (2) spinal or rachialgic ; (3) neuralgic ; (4) cardialgic ; (5) gastro-intestinal ; and (6) that connected with the generative organs. We shall propose a new division, and which although less complex, will embody a certain set of facts, hitherto, it appears to us, insufficiently differentiated, viz., local neurasthenia. It may suffice, we think, to divide neurasthenia into two forms according as to whether an excessive prevalence of one set of symptoms is absent or present. In this latter case we admit that such symptoms constitute a variety in relation to the localisation apparently revealed by it.

The following scheme explains this division :—

Neurasthenia.	Without an excessive predominance of any one set of symptoms.			General N.
	With such a predominance arising more particularly	from the central nervous system.	Cerebral.	Cerebral N.
			Spinal.	Spinal N.
			Sympathetic.	Sympathetic N.
		from the peripheral nervous system.		Local N.

(1) *General Neurasthenia.*—Under this category we shall place those cases in which the grouping of symptoms produces a uniform picture, but in such fashion that not one of them predominates over the others. These cases are in reality most common and widely distributed. We shall embody them into a concrete example, as a means of comparison. The subject is mostly a patient about 30 years of age suffering from the effects of moral distress or excessive work. He is wasted, pallid, and finds it difficult to get through his business. He feels weak and fatigued. He has almost continuous headache, and he indicates the nucha as the situation of it. Moreover, he experiences a crackling there. He fears lest it should be the beginning of some brain disease, the more so as sleep has often quite forsaken him. His appetite is diminished, his digestion slow, and he has a feeling of distension after food so that he has to loosen his dress. He has eructations and flushes of heat in the face. When asked, there is no particular organ in his body that he can refer his trouble to. He complains of pain at the heart and palpitation. Breathing requires an effort, the intestine is sluggish, and genital power diminished, &c. Yet objective examination reveals nothing more than a diminution of muscular power as made out by the dynamometer. Sensation is hardly affected, but

pressure on the lumbar spines is painful. There is nothing abnormal found in the special senses. The tendon reflexes are present. The pulse may be irregular and increased in frequency, but auscultation does not discover any sign of organic disease. Examination of the lungs is negative. As to the digestive organs, there may be a slight dilatation of the stomach, which, however, cannot be made responsible for the symptoms presented by the patient.

(2) *Cerebral Neurasthenia.*—In this, as in the following variety, the symptoms seem to arise more exclusively from the nervous system. The cerebral affection may assume a hemiplegic form (hemi-neurasthenia) as has been pointed out by Beard and confirmed by Prof. Charcot. In these cases the headache and weakening of the motor power is on the same side of the body. The various paræsthesiæ, sensations of tingling, cramps, &c., are equally unilateral. At times great difficulty of diagnosis results from these symptoms. Cerebral neurasthenia (cerebrasthenia) occurs mostly among the intellectual. It corresponds to overpressure in the ordinary sense of the word. It is more rarely attended by disturbance of nutrition in the shape of the wasting pallor and referred to above. The gastric symptoms are reduced to a minimum, and are absent in many cases. It is the mental fatigue which predominates, a sensation of emptiness in the head and an inaptitude for all intellectual work. Conversation itself soon becomes burdensome; hypochondriacal tendencies are almost always present. The character is modified, the patient is sad and preoccupied; he avoids society; he is extremely emotional and irritable. Moreover, headache, with its usual characters and situation, is constant. At times it reaches a degree of intensity so as to be able to make one fear a cerebral tumour. Insomnia is frequent, and sleep is disturbed by painful dreams. Finally vertigo completes the picture of the symptoms. At times this form of neurasthenia lasts long, and is rebellious to the varied methods of treatment.

(3) *Spinal Neurasthenia.*—This variety has been called myelasthenia. It corresponds to spinal irritation. It often follows upon venereal excess. It differs a little in man and

woman, although in both there is rachialgic pain and troubles in the reproductive system. In man one finds pain in the back, weakness in the lower extremities often accompanied by giving way of the legs. In addition, there is more or less complete impotence accompanied by spermatorrhœa. In the female the pain in the back radiates more frequently, and extends to the abdomen in the form of peri-uterine or ovarian pain. Pruritus vulvæ may exist. It is to this form that the type of disease wrongly known as neurasthenic pseudo-tabes ought to be referred. Pains more or less simulating lightning pains. Sometimes visceral crises, and a gait rather like that of vertigo than ataxia[1] are observed in these cases.

(4) *Sympathetic Neurasthenia.*—It will easily be understood that we should describe here cases in which there predominate troubles in one or other of the great systems, (1) cardiac, (2) gastric (cardialgic gastro-intestinal neurasthema of Pitres), and (3) respiratory (visceral neurasthenia of Huchard), as they partly draw their nerve supply from the sympathetic. We would include here a very interesting variety to which Professor Charcot has called attention, and where disturbances of nutrition prevail (trophic neurasthenia—might one say?).

Cardiac neurasthenia is typified in the neurasthenic angina pectoris of M. Huchard. This angina is benign, and has peculiar symptoms. The pain is less severe, its radiations less regular, and perhaps absent or appearing on both sides. The attack is often nocturnal, frequently repeated, and very often accompanied by palpitation and varied sensations. These cardialgic crises have a "blustering" behaviour, alternate with other symptoms, and have at times a frequency and periodicity, quite distinct from true angina.

It is to the cases in which symptoms connected with the digestive system prevail, that the "enteroptoses" of M. Glénard are to be referred. The principal symptoms, both functional and physical, related by this author are the following:—

1. Asthenic symptoms, debility, and habitual lassitude, weakness of the stomach, and of the kidneys.

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[1] See upon the subject of neurasthenic pseudo-tabes, Author's Review, *Gazette des hôpitaux*, 1890, p. 321.

2. Mesogastric symptoms (sensations of uneasiness, weight, emptiness, craving, sinking, dragging, and many other analogous morbid subjective feelings).

3. Gastric symptoms (flatulency, choking sensations, distension, oppression, wind, eructations, gaping, pain, acidity, cramp, burning, vomiting).

Both the asthenic and mesogastric symptoms present a close relation with the four following characters with which they are always associated.

1. Waking at 2 a.m., and after this *malaise*, and more or less sleeplessness.

2. Exacerbation or appearance of the *malaise* at rising in the morning, and at three o'clock in the afternoon.

3. Constant relation, marked or not, of discomfort according to character of the food (its aggravation by fats, starch, acids, vegetables, uncooked food, wine and milk.)

4. Irregularity and insufficiency of stools (constipation, diarrhœa, alternation of the conditions).

As to physical signs, methodical examination of the abdomen (mesogastrium), yield four important signs.

1. Flaccidity of abdomen (diminution of tension, "ventre en besace, gourde, bissac, bateau"); abdominal hypotasis.

2. Prolapsus, falling down of the intestinal mass, viz., enteroptosis, and as possible accessory signs, floating kidney (*nephroptosis*), moveable liver (*hepatoptosis*), moveable spleen (*splenoptosis*).

3. Narrowing of the colon ("boudin cæcal rénitent et sensible, cordon sigmoïdal, corde colique transverse," and as a consequence epigastic pulsations)—enterostenosis.

4. Gastric splashing, through prolapse and flaccidity of the stomach—gastroptosis and gastric atony.

In the paragraph concerning its pathogeny, we shall have to enquire whether the greater part of the cases of neurasthenia may be attributed to enteroptosis as M. Glénard pretends, or if, on the contrary, as we think, it only constitutes a single variety of the neurosis. It must be added that Glatz has proved, by examination, that the secretion of the gastric juice is arrested in a large number of the cases of gastric neurasthenia. In a case observed by us (the diagnosis being confirmed by Professor Charcot, and proved by the subsequent behaviour of the case) an analysis of the contents of the stomach by M. Durand-Fardel shewed an absence of hydrochloric acid, lactic acid, and peptones.

The variety called by us *trophic* neurasthenia presents this peculiarity which is characteristic of the case, viz., a condition of falling off in the patient almost amounting to cachexia. In these instances there is a general and marked weakness, a real wasting (the body weight diminishes, more or less rapidly in a notable proportion of cases), and a pallid earthy tint of the integuments. If we add to these signs certain more or less localised and painful phenomena, it will be readily seen that it may easily be mistaken for the cancerous cachexia. But the following facts are noted :—more or less rapid variations in the body weight, the preservation of the appetite, habitual absence of vomiting, and finally, a prolonged duration with the alternations already referred to.

(5) *Local neurasthenia.*—This form, which we have not found described by authors, at any rate not as a distinct type, might be called partial or dissociated neurasthenia. It is characterised by a single set of symptoms, although the patient suffers from hereditary taint, and from the mental condition peculiar to the neurasthenical. One might, we believe, legitimately compare this form to mono-symptomatic hysteria, where there exists but one symptom, contracture, vomiting, &c., with no other, and say that neurasthenia in such cases assumes a mono-symptomatic form.

By reason of the extreme variety of the subjective symptoms complained of by the neurasthenical, and because each of these symptoms may present itself alone, the aspects of this mono-symptomatic form are very numerous. The most characteristic is the one which I have proposed to call *topoalgia* (τοπος αλγη). Here there is pain fixed and localised in a region which does not correspond anatomically or physiologically to any recognised locality.

To this new class may be attached many cases wrongly classed under hysteria and hypochondriasis, when not left undetermined and relegated under the heading of "nervous symptoms." The symptoms are chiefly represented by pain. This appears in the form of patches—paræsthetic plaques—in very different parts, for instance, in the forehead, nucha, chest, præcordium, epigastrium, loins, buttocks, and upper and lower extremities. These painful areas are variable in extent and form, so that they do not lend themselves to description. Often the pain remains definitely localised in the particular region it has seized upon; at other times it may radiate from thence in different directions, and these radiations existing only at the time of the paroxysms, hardly ever attain the same degree of intensity as the fixed pain. The pain is continuous, disappearing only with sleep. It does not prevent sleep, but if the patient wakes up it returns in these wakeful intervals. It is not usually influenced by pressure exercised upon the body surface above it but it is liable to variations either spontaneous or brought about by stimuli, psychical or other. We have noted that it is generally not felt when the pain produced by faradisation is substituted.

The pain also presents paroxysmal crises returning with a certain periodicity and without cause, but at times called forth by physical *malaise*, intercurrent organic disease, or by emotion. It is to be remarked that the painful patch does not correspond in any sense with the distribution of a nerve, not any more than with the outlines of any organ. Moreover, objective examination carried out very attentively does not reveal anything abnormal. Yet in some cases a certain slight alteration in the cutaneous sensation corresponding more or less exactly to the area of the painful part, and consisting at times of hyperæsthesia or more frequently of hypæsthesia, may be observed. Moreover, there exists, and not very rarely, a vasomotor disturbance at the same spot consisting in an intense and rapid rubefaction of the skin under the influence of slight pressure.

It seems probable that a certain number of morbid conditions such as the glossodynia described by M. Verneuil, several forms of coccydynia, as also certain observations analogous to those reported by M. Galippe,[1] under the heading of "obsessions dentaires" may be included in this class, and considered as local neurastheniæ. We are also ready to classify as analogous forms of mono-symptomatic neurasthenia the instances of essential impotence, both psychical and spinal lately pointed out by us.[1] These local forms like their hysterical analogues are generally very obstinate.

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[1] Galippe. *Archives de Neurologie*, 1890.

(3) *Associations.*—Neurasthenia is associated in a variety of ways with a large number of other neuropathies, and particularly with hysteria, progressive locomotor ataxia, Graves' disease and general paralysis.

It may happen that one or other of these affections may develop in an already neurasthenical subject, or that neurasthenia shows itself at the same time as hysteria and exophthalmic goitre, or finally that it appears during the course of the myelopathy.

Among its most interesting relations are those with hysteria, especially with hysteria of traumatic origin. As already stated this question has been very fully elucidated in the later works of Professor Charcot. He has shown by the aid of clinical analysis that the supposed traumatic neurosis generally presents a variable mixture of neurasthenia and hysteria, which he has designated under the name of hystero-neurasthenia. It should be observed that previously to the full development of hystero-neurasthenia, a sort of premonitory stage, or incubation period so to speak, occurs. This is scarcely marked by anything, but slight neurasthenical symptoms. The hysteria appears then upon this neurasthenical basis, giving to this malady its well marked characters, but which, in spite of their combination may, nevertheless be referred each to its original neurosis.

The forms of neurasthenia which are most commonly combined with hysteria in these cases are the cerebral and spinal varieties—psychical phenomena, mental depression, sombre ideas, absence of will-power, excitability, insomnia, headache, enfeeblement of the sexual functions, seminal losses, and the sacral patch.

When neurasthenia is associated with tabes, it is unusual for the neurosis to precede the myelopathy. Generally it establishes itself when the latter has been developed for some time. In these cases it usually acquires the character of obstinate tenacity, and shows itself under a cerebral form, viz., in melancholic ideas, and hypochondriacal thoughts. Exophthalmic goitre is also often complicated with neurasthenia, but the co-existence of the two neuroses offers no noteworthy peculiarity.

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[1] Blocq. *Mercredi Médical*, 29 Octobre, 1890.

When neurasthenia precedes general paralysis, which is not a rare occurrence, it may through its own psychical symptoms mask the onset of the meningo-encephalitis, and open the way to unfortunate errors in diagnosis. Therefore, it is important to be forewarned of the possibility of this succession of events.

Progress, Duration, Termination.—As regards the course of the disease, an acute form has been described (Bouchut, Bouveret), to which indeed M. Huchard believes the phenomena of "nervous fever" properly so-called, should be attributed. We think that the observations alluded to here need much reserve. If acute neurasthenia exist, it is certainly very rare.

In the very great majority of cases, neurasthenia, even when following upon accident, commences slowly, and follows a continuous course with alternations of periods of aggravation and amelioration. This latter fact gives to the disease a sort of circular course (Déjerine[1]).

All cases are susceptible of improvement; most are completely curable, but others resist. Hereditary and obstinate neurasthenia may end in an incurable state of hypochondriasis. Moreover, the general debility of the neurasthenical makes them more vulnerable to external circumstances, which may prove fatal. It is necessary to know that neurasthenia, though apparently quite cured, may sometimes remain latent, only impressing its mark on whatever affection that may ultimately supervene.

Prognosis.—It will be seen from what has preceded that the prognosis as regards life is not serious, and that recovery is most often possible. However, neurasthenia is obstinate in resisting treatment, and prevents the patient from attending to his occupation; and it may even last indefinitely, all of which considerations diminish in some degree the favourableness of the prognosis.

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[1] DÉJERINE. *These d'agregation.*

From this point of view, neurasthenia may be divided into two classes, the primitive and the hereditary. In the case of the acquired neurasthenia, recovery is the rule. In the other case, where the neurasthenia is hereditary, and one might almost say allied to insanity, it is the most often incurable.

It should also be borne in mind that neurasthenia may lead to morphiomania and suicide, and that it constitutes a neuropathic taint capable of bringing about the development of various maladies in the descendants.

Diagnosis.—If it were necessary to deal here with all the diseases which have been, with more or less reason, mistaken for neurasthenia, we should have to review the greater part of both internal and external disease. We shall limit ourselves to mentioning those morbid processes which are more particularly apt to be mistaken for it. In the nervous affections of this kind, general paralysis and tumours of the brain must be referred to among cerebral diseases; locomotor ataxia among the spinal diseases, and hysteria and hypochondriasis among the neuroses. The mental depression, weakened memory, and melancholic tendencies, may suggest on-coming general paralysis. One is, up to a certain point, assured about this by the absence of any affection of the pupil, of difficulty in speech and of tremor; but it is often necessary to keep the patient under observation for some time, for the subsequent development of the case to settle the diagnosis.

Vertigo and headache might easily give the idea of a cerebral neoplasm, but the absence of eye changes, and the special characters of the headache in the two cases, will not permit of doubt for any length of time. If the neurasthenic patient be also syphilitic, the difficulty of diagnosis is greater, although the neurasthenic manifestations, both cerebral and gastro-intestinal, permit of avoiding error.

The proposed name of neurasthenic pseudo-tabes shows that a certain set of clinical manifestations have suggested the idea of tabes. The pains have not, however, the intensity, nor yet the fulguvating character of those met with in locomotor ataxia. The knee jerks are not abolished, nor are ocular troubles to be made out, nor do the visceral phenomena resemble, in any degree, the visceral crises of that affection.

Hysteria, even when associated with neurasthenia, may be distinguished by the convulsive seizures, the presence of stigmata, sensitivo-sensorial hemianæsthesia or segmental anæsthesia, hysterogenetic zones, diminution of the field of vision, and anæsthesia of the palate.

The hypochondriasis of insanity is differentiated by the fixity and intensity of the morbid ideas and the complete inability to displace them by any kind of reasoning.

M. Huchard has asked whether neurasthenia should be diagnosed in that form of rheumatism which is known as vague or neurotic rheumatism. The diagnosis, he continues, consists rather in comparison, for we are convinced that the two maladies are nearly always confounded, and that they form one and the same morbid condition. The gastric troubles of neurasthenia may cause the neurosis to be mistaken for organic disease of the stomach, gastritis or cancer. The error is so much the more to be feared, inasmuch as examination often shows, as has been already pointed out, a diminution or even absence of the gastric juice, as in the true gastropathies. The diagnosis is then based upon the absence of vomiting in neurasthenia, upon the duration of the disease, upon the fluctuation between improvement and aggravation, and finally upon the absence of a tumour or glandular affection. We shall not return to the signs which permit of the diagnosis being made between true angina pectoris and the neurasthenic form of the disease, for they have already been pointed out.

Nature and pathogeny.—Neurasthenia has not in reality any pathological anatomy proper to it, for it cannot be granted that it depends on any such anatomical substratum as the lesion in the digestive tract advocated by M. Glénard. These lesions, even supposing them capable of producing the symptoms of neurasthenia, a possibility we are about to examine, would, at any rate, only act indirectly by changes in the general condition, and consequently in the nervous system in particular, brought about by them.

Thus failing a firm basis of necroscopical proof for building up a pathogeny, we shall only indicate, and that with reserve, the hypothesis justified by the clinical aspects of this affection.

In the first place we think with Professor Charcot[1] that neurasthenia is a morbid state, the nosographical position of which cannot be disputed, since it is seen to preserve its individuality through the very varying conditions under which it arises. It seems to us equally true that the statement of M. Huchard, that in the majority of cases neurasthenia is an arthritic neurosis, cannot be applied to it. Finally, we believe that in spite of the variety of its appearances (which latter alone have served as a guide in the clinical classification proposed here), neurasthenia is a neurosis essentially localised in the brain. Its characteristic is the enfeeblement of energy in the various cerebral functions.

The subjects affected with neurasthenia are generally proof against suggestion, especially if they be compared with the hysterical, in whom the aptitude for suggestion is one of the fundamental characteristics of the mental state. Hysterical patients have a perversion of the will, the neurasthenical a "catalepsy" of the will, following the expression of M. Huchard. Whence this difference, capable, in our opinion, of explaining the psychology of neurasthenia? And this is a matter of capital importance in the question of the pathogeny of the disease.

In the hysterical individual, it is as a result of a sort of limitation in the field of consciousness that the idea acquires an intensity which makes it dominant. It is in relation to this concentration of the conscient activity that the other brain-functions appear to be depressed when they are only inactive. The neurasthenical, on the other hand, is unable to generate a strong idea in consequence of the real depression of all the parts of his intellect. If he perceives, and more particularly if he preserves for long, certain sensitive images, which are more or less intense and which take possession of him, it is the result of the abnormal activity of ordinarily automatic and silent mechanisms presiding over the organic functions.

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[1] Charcot. *Policlinique*, 1888-1889, 12e leçon, p. 260.

This enfeeblement, localised chiefly in the intellectual centres, determines the cerebral form of neurasthenia. The diminution of the regulating power exercised normally by the brain over the other parts of the nervous system, the spinal cord, and sympathetic, explains the existence of the spinal and sympathetic form of the disease. The visceral functions especially, usually performed unconsciously, enter into the sphere of the patient's consciousness, giving rise to a whole series of abnormal sensations.

As to the local forms of neurasthenia, and particularly topoalgia we think that they represent the clinical manifestations of a persistent and fixed sensory image, analogous in the domain of sensation to what the fixed idea is in that of the intellect. The mechanism of this manifestation differs from that of the auto-suggestions of pain belonging to hysteria, and of the emotional prepossessions of the hypochondriacal.

What is the relationship between this neurosis and enteroptosis, according to this mode of looking at the nature of neurasthenia?

The fact of the co-existence of visceral prolapse with the nervous phenomena and the disappearance of the signs of neurasthenia in consequence of the treatment of splanchnoptosis appear to be strongly in favour of the subordination of the one to the other. Enteroptosis would act according to M. Glénard, in bringing about a lessening of the lumen, or atresia, of the bowel, and in consequence gastric atony.

Without denying the relations of enteroptosis and neurasthenia, it does not seem admissible that the meso-gastric troubles should be considered as specific ætiological elements of the neurosis. In the first place it is very frequent to see neurasthenic patients in whom there are none of the objective signs of M. Glénard, and in whom even gastric symptoms are very little complained of. Moreover, certain forms of neurasthema are developed more or less rapidly as a consequence of mental overstrain, of emotions, of traumatism, all of them causes which do not usually produce mechanical intestinal disorders. Hereditary influence, finally, is often undoubted from the point of view of pathogeny, and moreover, a favourable termination without treatment directed to the intestinal tract is not uncommon.

It is rather through the general disorders of nutrition from gastric atony (and which are the least doubted consequences of enteroptosis) that this ailment plays its ætiological *rôle* in the production of neurasthenia—a *rôle* reduced thus to that of an exciting agent.

The neurasthenic state is the first step of the neuropathic ladder, and "a soil eminently favourable to the development of nervous disease in future generations" (Bouveret). This idea, which has been especially developed by Mœbius,[1] has afterwards been taken up, and adopted by M. Déjerine.[2]

Treatment.—Before explaining the rules of treatment suitable to the varied forms of neurasthenia, it is necessary to formulate certain considerations of a general kind.

Not only does this malady offer considerable resistance to medicinal treatment, but what is more, these patients exhibit idiosyncracies to drugs. In some cases therapeutical agents do not produce their usual effects; and in other cases they bring about exceptional results. "In a word, one often has to deal," as M. Huchard says, "with a veritable therapeutic ataxia, since in neurasthenia, that which should react, viz., the organism, either does not react or reacts abnormally to chemical and medicinal substances." If then it is necessary to guard oneself against the abuse of drugs, it is not less necessary, in order to secure the success of the treatment, to inspire confidence in the patient, and to exercise over him considerable authority.

For this purpose, it is undoubtedly proper to ask oneself how far hypnotism and suggestion are indicated.

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[1] Mœbius. *Ueber Nervöse Familien.* Allg. Zeitschr. f. Psych., Berlin, 1884.

[2] *L'hérédité dans les maladies du systemè nerveux.* *Thesis* 1886, pp. 170, et 226.

There is no better test example, in our opinion, than that of neurasthenia, to show the limits of hypnosis from a therapeutical point of view. In fact, would it not appear, *a priori*, that in this psychical and dynamical affection, hypnotic suggestion should work wonders? In reality there is nothing to be expected from it. Very few neurasthenic patients are hypnotisable, according to the statement of even Professor Bernheim.[1] The mental state of the neurasthenic patient such as we have described, appears to account for this fact of observation. It ought to be stated, however, that M. Bernheim has reported some examples of cure of affections which he terms "neurasthenic;" but in the majority of these cases it appears as if neurasthenia properly so-called were out of the question.

We think that hypnotic suggestion cannot figure among the usual therapeutic methods of the treatment of neurasthenia, and that it can scarcely be of service unless in exceptional cases. In the ordinary and benign form of neurasthenia, hydrotherapeutic treatment associated with the use of the actual cautery and of bromide of potassium is generally sufficient.

In the severe forms the Weir Mitchell method may be had recourse to with success. It consists as is well-known, in isolation, absolute repose, massage, faradisation, and over-feeding. In the cases in which the presence of enteroptosis is proved, one may rightly make use of the method employed successfully by M. Glénard. Succinctly stated, it consists of (1) the use of the pelvic belt, day and night; (2) regulation of the bowels—a daily laxative; (3) feeding—including four meals. A bread-soup, or café au lait, in the morning. A meal of meat consisting of roast beef or mutton; and a boiled egg with bread at 11 a.m.; a repast at 4 p.m. with bread, jam and tea. At 6.30 p.m., a meal like that at 11 a.m. Beer or alkaline water is taken as drink; (4) alkalinisation—sodium bi-carbonate or Vichy water; (5) hydrotherapy of the form of cold douching, or 20-30 seconds.

In the local mono-symptomatic forms we have used with success, faradisation localised to the seat of the painful phenomena by means of the electric brush.

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[1] BERNHEIM. *Hypnotism, Suggestion, Psychotherapie,* Paris, 1891.

We may add the exhibition of the fluid extract of kola, suggested by M. Huchard, and the employment of static electricity, often prescribed by M. Charcot, in order to complete the list of the therapeutical measures most commonly used, either isolated or combined, with benefit to the patient.

Reviews and Notices of Books.

Ueber Multiple Neuritis. Von Dr. J. Pal. *Wiener Klinischen Wochenschrift.*

Eight original cases are detailed in this paper. They illustrate, on the anatomical side, the changes that may be found in the spinal cord in cases of multiple neuritis, such as degeneration of the tracts of white matter, or small hæmorrhages into the cord. These changes (in the author's view) are related to the neuritis not either as cause or as effect, but are probably produced by the action of the same cause that produced the neuritis. On the clinical side this question of causation is illustrated by the author's cases: two of them were referable to the commonest cause, alcohol; one to lead-poisoning; one to arsenical poisoning; two to acute febrile diseases, viz., erysipelas and typhus fever; two were cases of acute multiple neuritis, whose causation was uncertain.

In all these cases the neuritis appeared to be primary, and not of the nature of degeneration secondary to disease of the anterior cornual cells of the cord, for these cells were found to be practically healthy.

Case I.—Man, aged twenty-seven, of good previous history and temperate habits. Illness of eleven days' duration on admission, following a chill, and consisting of pains in the legs, then in the arms, difficulty of speech, distortion of the face, profuse sweating, constipation, difficulty of walking. The patient walked with a staggering, dragging gait; the knee-jerks were diminished, the nerve trunks tender to pressure, but not the muscles; there was slight œdema of the feet, the left sixth nerve was paralysed, and the right facial. The paralysis of the limbs, especially of their extensor muscles, increased rapidly, the knee-jerks disappeared, and about twenty days from the onset œdema of the lungs developed and proved fatal. The temperature was throughout normal, in spite of the rapid course of the disease. Shortly before death the muscles were tested with the faradic current; the farado-contractility was diminished, though present; the muscle-twitch was sluggish.

After death the right vagus, both phrenics, and right sciatic nerves were found to be degenerated (breaking up of the myelin). In the central nervous system was found capillary hyperæmia of the grey substance of the cord, medulla, and cerebellum, numerous capillary hæmorrhages in the posterior horn of the lumbar cord, and the anterior horn (right) of the cervical cord (segments 3 to 6). There were microscopical hæmorrhages in some of the spinal ganglia.

The author regards this as a typical case of a somewhat rare disease, viz., acute multiple neuritis. Its causation was obscure; the possibility of infection with micro-organisms could not be excluded, since all the nervous system was hardened in Müller's fluid. He remarks upon the clinical resemblance to Landry's disease, and considers that though Landry's disease may be possibly due to multiple neuritis, it has not yet been proved that this is always the case.

Case II.—Subacute relapsing multiple neuritis associated with pulmonary and intestinal phthisis, in an intemperate man of twenty-eight years old. The exciting cause of the original attack was said to be a chill. Parenchymatous degeneration of several nerve trunks was found microscopically. In the cord there was found: (*a*) lumbar region—partial degeneration of Lissauer's zone; (β) cervical region—degeneration of Goll's tracts, consisting of great thickening of the neuroglia, with slight affection of the nerve fibres. There was no disease of the posterior nerve-roots in the cervical region, though in the posterior roots of the cauda equina there appears to have been some doubtful neuritis.

Case III. resembles Case II. so far as the clinical history is concerned. There was in addition caries of the spine (cervico-dorsal region). Degeneration of peripheral nerves was found, and in the spinal cord (level of second and third cervical segment) marked degeneration of Goll's column, and of a point on the periphery of each anterior column (near the place of egress of the anterior nerve-roots). The author is not inclined to view this as a secondary degeneration started by the cervical caries, because at the level of the caries the cord was normal.

Case IV.—An anomalous case of lead-poisoning, in which degenerations both of the peripheral nerves and of the spinal cord were found *post-mortem*.

Female, aged twenty-one, compositor; chronic lead poisoning for two years previous to admission, the symptoms being chiefly, paralysis of left sixth and facial nerves, pallor of the optic discs, vomiting and colic. From these she had recovered; on admission in August, 1890, the chief symptoms were tremor, motor weakness, and stupor; subsequently there developed, in September, amaurosis from optic neuritis, recurrence of paralysis in the cranial nerves; in October, ataxia of upper limbs, delusions (she thought her hands and feet were cut off), there was ankle clonus and patellar clonus; later (November) there was absence of patellar tendon reflex, wasting of the limbs, bilateral paralysis of the peronei, œdema of the feet, bedsores; and she died in this condition.

The following parts were subjected to microscopic examination:—

(1) Nerves: the optic, oculo-motor, sixth, facial, auditory, radial of right side, the peroneal of left side. All these showed degeneration, except the auditory.

(2) Spinal cord and nerve roots.

In the cord were degenerations of the white matter, both in the posterior and lateral columns, which may be epitomized as follows:—

a. Posterior columns and posterior nerve-roots. Degeneration of postero-lateral columns, beginning at the mid-dorsal region, and extending upwards through the upper dorsal and cervical regions.

This was probably secondary to disease of the posterior nerve-roots, which were degenerated throughout the cervical region (and also in the upper half of the dorsal region ?). After entering the cord, only those fibres which go to the posterior columns were diseased, not those going to Lissauer's tracts.

In the postero-median columns, extending over the same length of cord, were two small degenerate areas, one on each side, that on the right side being most extensive, situate at the posterior part of the column.

With reference to the disease of the nerve-roots, it should be added that one of the ganglia on these roots was examined and found normal, the disease beginning in the nerve-root between it and the cord.

β. The antero-lateral columns showed rather more complicated changes.

In the upper dorsal region there was degeneration involving the cerebellar tracts, and the crossed pyramidal tracts (most marked on left side of cord), and further a small spot of degeneration in each direct pyramidal tract.

Above this (cervical fifth) the cerebellar tracts were degenerate, but the disease spread from them slightly inwards towards the grey matter. Still further up (cervical second) the disease was fairly well limited to the cerebellar tracts.

Below the upper dorsal region the degeneration of the lateral columns became more and more limited to the crossed pyramidal tracts, which in the lumbar region were the only parts diseased.

As the author remarks, these degenerations in the antero-lateral columns were such as might be secondary to a focus of diffuse myelitis in the upper dorsal region, but no such focus of myelitis could be found.

We must therefore suppose that in this case there was not only disease of the nerves, but also combined tract disease of the cord—disease of the posterior columns and posterior nerve roots of the cervical region (tabes cervicalis), disease of the cerebellar tracts, and in the lower half of the cord of the descending pyramidal tracts.

The remaining cases 'which were cured' are cited for points of clinical interest.

Case V.—A woman, aged thirty-four, had erysipelas of the trunk after a surgical operation. This was followed by paralysis of the following nature: paraplegia with absence of patellar tendon reflex, contraction of flexors of the knees, flabbiness and wasting of other muscles; in the upper limbs, extensor paralysis and muscular wasting; tenderness of nerve trunks to pressure; electrical changes even in parts that were not paralysed; a complete loss of memory, apparently resembling that of alcoholic neuritis, though there was no history of alcoholism. Recovery under treatment by massage. The patient had had a similar attack nine years ago after erysipelas of the face. Hence it seems that the erysipelas was the veritable source of the neuritis, though such a sequence of events is extremely rare.

Case VI.—Paraplegia, due to peripheral neuritis, following an attack of typhus, and recovering under massage. There was a peculiar condition of plantar reflex, the limb responding to a single irritation not once only, but twice or even three times.

In both Cases V. and VI. the cutaneous sensibility for the faradic current was impaired out of all proportion to the loss of tactile sensibility.

Case VII.—Acute multiple neuritis in a man aged thirty-one, of uncertain causation, presenting the following features: hyperæsthesia of skin, extreme tenderness of muscles (so great as to suggest a primary myositis), subsequent tenderness of nerve trunks; extensor palsy of upper limbs; while in the lower limbs there was preservation of patellar tendon reflex, although the extensor muscles of the knees showed reaction of degeneration. Recovery.

Case VIII.—Peripheral neuritis after acute arsenical poisoning; eleven months' duration on admission. A tolerable recovery was made, although some muscles were so deeply affected as to have lost all electrical irritability.

The author gives the following general conclusions on the subject:—

(1) Multiple neuritis frequently occurs as a primary affection, independently of any disease of the anterior cornual cells of the cord.

(2) In the course of a multiple neuritis, lesions of the central nervous system may take place (*e.g.*, capillary hæmorrhages), evoked by the same cause that produced the neuritis.

(3) The strands of white matter, *e.g.*, Goll's columns, Burdach's columns, &c., &c., may be degenerated; the degeneration in such cases need not be strictly "systematic" in distribution.

(4) As to "latent neuritis," particularly the neuritis that is said to occur in tubercular subjects, the author thinks that œdema of the feet (cachectic œdema) and diminution of the cutaneous sensibility to faradic currents, are often the only symptoms of such neuritis.

J. A. Ormerod, M.D.

Du Vertige Cardio-Vasculaire ou Vertige des Arterio-Sclereux. Par le Professeur J. Grasset. Paris: Georges Masson, 1890.

Probably any one who will devote a few hours to the study of the eighty pages composing this volume will find their interest in the subject of vertigo agreeably quickened thereby.

Professor Grasset first devotes his attention to the subject of vertigo in general, and gives it as his opinion that it is a sensation, a sensation of loss of equilibrium. It is accompanied by a certain amount of fear, and may cause the patient to fall; there are also certain sensory phenomena affecting the sight and the hearing: but there is not as a general rule any loss of consciousness. Some gastric troubles, as nausea and vomiting, complete the picture; and the Professor insists, as we think rightly, that it is wrong to call vertigo with some ear symptoms Menière's disease, or to refer vertigo, which is accompanied by nausea and vomiting, to the stomach. Some such troubles are concomitant symptoms of all vertigo, and their mere presence in any case is not sufficient to prove its origin. Severe vertigo borders on syncope on the one hand, or epilepliform attacks on the other.

The effect of opening or shutting the eyes serves to distinguish certain kinds of vertigo. Thus the vertigo of sea-sickness or that of open spaces or altitudes improves or ceases entirely when the eyes are closed; while, on the other hand, the vertigo of indigestion or alcohol may only come on when the eyes are shut. With this latter group he places the sign of Romberg in ataxy, holding that it comes within the definition of vertigo and is not due to plantar anæsthesia.

He then proceeds to divide vertigo into (*a*) acute or accidental, which occurs only once or occasionally and is of little real importance—such as the vertigo of acute diseases, that of dyspepsia, acute alcoholism, and certain other toxic states; and (*b*) chronic or habitual vertigo, which again is sub-divided into (1) epileptic vertigo, (2) sensory vertigo—including Menière's disease, and (3) stomachal vertigo; and to these he presently adds (4) cardio-vascular vertigo or the vertigo of arterial sclerosis, the special subject of these lessons.

By arterial sclerosis is meant an arteritis, a mixture in variable proportions of endarteritis and periarteritis; it affects all the arteries of the body, but tends to produce its most serious results in certain localities, as the heart, the kidney, the cord, or the brain. The first stage is thus one of general arterial change, the second stage is this, plus the symptoms of the organ whose nutrition and function are specially affected.

It is found in association with gout, rheumatism, diabetes, asthma, migraine, lithiasis, and obesity. Chronic intoxication, with alcohol, lead, or tobacco, produces it, as also malaria or syphilis; and, to a less extent, acute infective diseases; hard work and old age also contribute something towards the result, which is generally due to the mixed action of several causes.

According to Huchard, whom the author quotes, the first stage of all is a spasm of arterioles, which, becoming gradually more persistent, produces high arterial tension, which in turn is the cause of arteritis and arterio-sclerosis.

This explains the important fact that in their early stages the phenomena of arterio-sclerosis are intermittent, and can be affected by remedies, while later on they are permanent and curable only with difficulty.

The symptoms of high arterial tension, such as dyspnœa on exertion, local syncope, &c., are mentioned, and to these are added cephalalgia, somnolence and vertigo, the special signs of deficient cerebral irrigation; while among physical signs the most important is a loud second sound over the aortic area of the heart.

A series of very interesting cases in illustration of these points form the major part of the last half of the volume, and a few remarks on the treatment of the various forms of vertigo and of the cardio-vascular form in particular in the last three pages conclude the work.

For epileptic vertigo bromide of sodium in large dose long continued is recommended, for Menière's disease sulphate of quinine, and for stomachal vertigo the treatment of the dyspepsia.

The final conclusion is that arterial sclerosis when it specially affects the nerve centres is capable of producing the three degrees of vertigo, (1) simple vertigo, (2) vertigo with some epileptiform attacks, and (3) vertigo with permanently slow pulse, accompanied by attacks of syncope or epilepsy.

The treatment of these conditions is the treatment of the arterio-sclerosis, or, rather, of the high arterial tension, which is the prime cause of both functional and organic changes; and the drugs on the use of which most stress is laid are iodide of sodium and trinitrin. They may be either taken simultaneously or alternately, and should be continued for months.

It is also necessary to cut off entirely tobacco, alcohol, strong meats and spices, and to recommend life in the open air, milk food, white meat and vegetables.

We have here then, it will be seen, not only an extremely able contribution, to our knowledge, of the etiology of vertigo in general, but also an interesting explanation of the way in which high arterial tension (or may we suggest the cause of high arterial tension, which is not mentioned?) may produce vertigo by bringing about temporary constriction of the arterioles of portions of the nerve centres. This enables us at once to see why vertigo should be classed with the paroxysmal neuroses, such as epilepsy or migraine, or border on syncope, because a similar constriction of arterioles is probably in each case the occasioning cause.

But be this as it may, we can cordially recommend this little volume as putting forward in a most interesting and instructive manner many points which it is important to bear in mind in the diagnosis and treatment of vertigo.

A. Haig, M.D.

International Clinics: a quarterly of Clinical Lectures. Young J. Pentland, Edinburgh and London.

The aim of the editors is "to make this periodical," which is to appear quarterly, "a complete post-graduate course of medical instruction," and the first volume has certainly some claim to be considered, so far as it goes, a fairly successful attempt in this direction. It is a collection of recent clinical lectures given by a portion of presumably "the most prominent teachers of all the well-known colleges of medicine and hospitals of the United States, Great Britain, and Canada;" and we notice that the Transatlantic section of this powerful combination of men of light and leading in the medical world contribute some three-fourths of the lectures contained in the present volume, a proportion which is hardly calculated to secure it on that side of the water where it is supposed to appear, that quantum of support which maketh glad the heart of the publisher.

The complete series of lectures are apparently intended to embrace the clinical side of every subject within the whole range of the healing art, so that when complete the series now initiated will be of itself an exhaustive clinical encyclopædia of every known disease.

The present issue touches upon many subjects of interest to the neurologist, the first lecture being on a case of acromegaly, by Prof. James Ross, of Manchester. Several tables of measurements in every conceivable direction are given, though it seems a little uncertain with what normal standard these are to be compared. There are also perimetric tracings of the patient's field of vision, which is deficient bi-temporally, and some reproductions of photographs of the patient. In this case Dr. Ross has reason to think there is persistence of the thymus gland, and apparently he attributes the state of the field of vision to pressure on the optic chiasma by a possibly enlarged pituitary body. He believes with Erb that the nearest allies of this disease will be found to be myxœdema and sporadic cretinism, and that the alteration in the bones will prove to be secondary to the morbid alterations in the ductless glands.

Prof. Shaltuck, of Harvard, contributes lectures on a case of uræmic convulsions and on a case of epilepsy, both of a type calculated to appeal to the student rather than the practitioner.

Mr. Hulke, of the Middlesex Hospital, gives a careful account of an interesting case of cranial injury in a woman with a syphilitic taint, judicially tracing back the symptoms to the most probable traumatic lesions.

Prof. Parvin, of Jefferson Medical College, in discussing a "Case of Ovaralgia with Spinal Irritation and Anæmia," devotes himself mainly to a consideration of the immorality of oöphorectomy, or of a pretended performance of this operation in the treatment of such cases.

Prof. Gray, of New York, is represented by a short lecture of an elementary character on "Different types of Paralysis in Young Children," differentiating them into two groups, cerebral and spinal. Myopathies he does not mention.

Sir Dyce Duckworth contributes an interesting lecture on chorea, illustrated by four cases. He regards it as a symptom, not as a disease, attempting to prove the thesis that chorea is a manifestation of the rheumatic habit or diathesis, which is in itself primarily a disease of the nervous system, and introducing the hypothesis of a peculiar neurosis—capable of hereditary transmission or transformation in the subjects of which may be set up at one time rheumatic, at another choreic, manifestations—to explain the many cases of chorea in which no rheumatic history can be obtained. Chloral is given the palm as controlling the movements, and arsenic as restoring to the normal the perverted nutrition of the nerve centres.

Prof. Wood, of the University of Pennsylvania, gives two good lectures on "Traumalic sore back" and "Traumalic hysteria" respectively, describing in the latter the successful result of a varied treatment, too heroic in some of its details, we are afraid, to be submitted to by patients here.

Prof. Sachs, of New York, sends over a lecture on "Neurasthenia," illustrated by four cases, interesting in their way, in which he recommends the usual routine treatment.

Prof. Mills, of Philadelphia, contributes a really good lecture on two very interesting cases of myotonia (Thomsen's disease) and athetoid spasm respectively. Though in the former the electrical reaction which Erb considers peculiar to the disease was not elicited, yet in other respects the case seems to have been typical, and the consideration of it is well worth reading.

Prof. David Ferrier is represented by a lecture of the typical post-graduate order on "Alcoholic paralysis," in which considerable attention is devoted to its pathology. Antipyrin is recommended to relieve the pains and paræsthesiæ.

Dr. Weir Mitchell gives a very useful and suggestive lecture on "The Treatment of Obstinate Sciatic Pain by Splint Rest and Cold," a method which he claims has succeeded in most obstinate cases of sciatic neuritis amenable to no other treatment.

Prof. Julian Chisholm, of the University of Maryland, contributes a useful and lucid lecture on "Astigmatism, a very common and often unrecognised cause of Headache," and certainly his advice to the general practitioner to constantly bear in mind the *raison d'être* of his lecture is not uncalled for.

The general substance of the lectures seems of a kind to render them more fitted for senior students than for recently qualified practitioners, and to the former we can recommend them as embracing many essentials only lightly touched upon in text-books.

E. A. Shaw.

"*Die Myoclonie.*" By Prof. Dr. H. Unverricht, Director der Medicinischer Klinik zu Dorpat.

This is a valuable addition to the literature on the subject, for the author has been at great pains to collect all the previously recorded cases, and to weed out from the literature those conditions which have been confounded with that under consideration. An attempt has been made to classify the cases into certain distinct groups, the wisdom of which attempt we are inclined to question, for in the present state of our knowledge, the result must necessarily be very imperfect, as is the case here. Several new cases are recorded, otherwise the work cannot be said to have added many new facts to our previous knowledge of the subject. The part which deals with the differential diagnosis deserves more than passing notice, as it has been dealt with in a masterly manner.

Allusion is first made to Friedreich, as having singled out this affection from the chaos of motor neuroses, and as having shown that it is a distinct affection, presenting a well-marked, and easily recognisable clinical picture. The symptoms which Friedreich considered typical of the condition, and his views as to its nature, are then referred to. The cases of five sisters, who came under the observation of the author, are next quoted in full, and the symptoms which characterise this clinical entity discussed *seriatim*, after which the conditions with which it is most likely to be confounded are mentioned, and the points which serve to distinguish them, pointed out.

We fully concur with the author when, after referring to the condition described by Hammond under the term "Convulsive Tremor," he questions the advisability of attempting, as the

American physicians have done, to recognise several distinct affections; in the present state of our knowledge of the subject, as it is far better to try to keep together all those conditions whose close relationship bring them under a common type.

The fourth section of the work reviews the literature, and collects all formerly recorded cases into five groups. The first includes all those which resemble his own and Friedreich's, the characteristic features of which are given, and attention called to the difference in the state of the reflexes in Friedreich's cases, in which they were exalted, and their condition in all other recorded cases, in which they were unaltered. The second group includes cases closely resembling those of the first, but presenting, in addition, features which liken them to other neuroses, such as Chorea, Hysteria, and the like. The third and fourth groups include cases like Chorea and Hysteria respectively, while the fifth includes two cases, one resembling "Paramyoclonus" and the other "Chorée rhythmée" (Charcot). The ætiology and pathology are discussed in the next section, and it is shown that whereas Friedreich attributed the condition to fright, this element was out of the question in the author's cases, which must be looked upon as "congenital myoclonus," or more strictly speaking "family myoclonus." After referring to the non-discovery of any anatomical lesion, he proceeds to give the various views that have been put forward as to the probable pathology, and his reasons for rejecting or supporting them. Papow's view that the defect is in the muscles themselves, is very properly rejected, that author's argument in support of his theory being quite untenable. We are also at one with him in considering Minkowski's arguments, in support of its being due to a cortical defect, based upon a misconception of the physiology of the cerebral and spinal centres, apart from the fact that his were probably not cases of myoclonus at all. Gravitz's cortical theory is negatived, chiefly owing to the symmetry of the affection of individual muscles on the two sides of the body. Friedreich's original theory, that the motor ganglion cells in the anterior horns of grey matter of the spinal cord are at fault, is accepted as the most plausible; but whether they are primarily or reflexly affected, the author does not pretend to say.

The prognosis is considered, though it is admitted that we are not yet in a position to be too definite on this point.

As to treatment, chloral, bromide and the voltaic current are all mentioned as being worthy of a trial, most reliance being placed on the first of these.

L'Hystérie Male. Par le Dr. EMILE BITOT, Chef de Clinique Medicale à la Faculté de Médecine de Bordeaux.

THIS work does not call for much comment, as the author does not pretend to aim very high, and has consequently not laid himself open to much criticism. All that is aimed at is attained, and that is the most we can say for the work, for in the introduction it is clearly stated that the work is not meant to be a complete history and review of male hysteria, but rather a work of observation, in which it is proposed to analyse the twenty-two cases which form the basis of the work, and to see whether they agree with or differ from the established rules.

The cases are divided into two groups, according as the affection is paralytic or spasmodic. The first chapter treats of hysterical paralysis in general, and, in reviewing the history of the subject, it is admitted that foreign schools have contributed new and important facts to the literature, but for the French school is claimed, with reason, the distinction of having analysed and classified the various forms of the affection ; and, of course, to Charcot is ascribed the highest honour in this connection. The characters of the various forms of hysterical paralysis are then gone into, and in speaking of the hemiplegic form, the assertion with regard to the absolute integrity of the face is objected to, as, although not paralysed, it is subject to various modifications in the form of spasmodic affections.

The author's paralytic cases are quoted in full, and their leading symptoms discussed. The second chapter deals with the spasmodic affections. Here the division of tremors by Charcot is alluded to, in which those of hysteria are placed, in point of rate of oscillations, between those of conditions such as Paralysis Agitans (four to five per second) and the more rapid ones of conditions such as Exophthalmic Goitre (eight or nine per second). Pitres' views on this subject, and his classification of hysterical tremors into trepidational, vibratory and intentional are next mentioned, and the author goes on to point out how such tremors can be diagnosed from those due to organic diseases, by certain modifying agents, *e.g.*, electricity, which has no effect on the organic, whereas it, as a rule, causes the functional to become exaggerated. The spasmodic cases are recorded and discussed, and the complete series of twenty-two cases tabulated. In reviewing the table, attention is called to the fact there brought out, that hysterical troubles increase up to the age of forty years, and other observers' statistics are quoted in confirmation of this. It is further pointed out that the old idea that hysteria is only met with in the luxurious classes is not borne out, as all the cases recorded in this work are those of hard-worked people, who had good constitutions, and who were strong and robust.

The part which heredity plays in the ætiology of such affections is referred to; and of the exciting causes, the first place is given to traumatism, emotion and intoxication by lead, mercury and alcohol. In conclusion, tables of comparison between male and female hysteria are drawn up, and traumatism is shown to be the most potent cause in males, while moral emotion is by far the most common cause in females. It is further shown that while men exhibit an excess of the paralytic and spasmodic affections, women exhibit an excess when all hysterical conditions are considered as a whole, for the condition is then found to be twice as common in females.

J. S. RISIEN RUSSELL.

Flushing and Morbid Blushing. By HARRY CAMPBELL, M.D., B.S.Lond. London: H. K. Lewis.

DR. CAMPBELL'S book deals with interesting if still somewhat obscure conditions. In the present state of our knowledge of the vaso-motor system and the manner in which its functions are performed, it is not to be expected that any explanation of such phenomena as are here discussed can be either full or final in its details. But in this volume a large mass of clinical material is used, and an attempt, interesting and suggestive, is made to furnish an explanation of the various conditions. First of all a succinct account is given of what may be called the Jacksonian conception of the nervous system, as divided into three evolutionary levels. With this as a background the subject of flushing is next discussed in its different manifestations, and the view is propounded that in this condition there is what is called a "nerve storm," a discharge of nervous energy, and in view of the fact that this discharge is accompanied by emotional and intellectual states, it is supposed to originate in the highest evolutionary level.

Morbid blushing is regarded as closely analogous, differing, however, in being as a rule determined by a mental state, and in having manifest vaso-motor phenomena; while in flushing, on the other hand, there *may* be only subjective sensations of vaso-motor changes, without any objective phenomena. In both cases it will be seen the vaso-motor conditions are regarded as secondary. Such a view necessarily supposes a condition of cells more or less unstable, and this leads to a discussion of the pathology of this and other neuroses. A good deal is said about the plasmic environment of the cell, and if in this part the writing is discursive and a trifle didactic, it cannot be said to be dull. As regards treatment, special stress is laid on good hygienic surroundings, and as to blushing in particular, on education carried out with the view of discouraging premature and excessive introspection.

The theory, it will be seen, is novel and ingenious, and the book is one which will be read with interest.

James Taylor, M.D.

Abstracts of British and Foreign Journals.

A Case of Tabes Dorsalis with Syphilitic Cerebro-spinal Meningitis. By Sidney Kuh, of Chicago. With a Lithographed Plate.

The author reports a case in which a typical tabetic degeneration of the spinal cord co-existed with syphilitic cerebro-spinal meningitis. H. W., an actor, first came under observation in December, 1887, the first symptoms of his disease appearing at that time. At the age of twenty he had a chancre, but no secondary symptoms. He attributed his illness to exposure to cold and overwork. Professor Erb made the diagnosis of incipient tabes dorsalis. The patient was a large powerfully built man; he had no lightning pains, but occasionally suffered from cramps in the legs; loss of sexual power for one year; sphincters intact; pupils large, unequal, re-acting only slowly to light; knee-jerks absent; no strabismus; sensation, especially to pain, deficient over the toes. Romberg's symptom slightly marked.

Under active mercurial and electrical treatment the patient's state had much improved by May, 1888. At the end of July, however, he was much worse; the defect of sensation had reached a considerable degree over the feet, and extended to the legs; he walked with difficulty and ataxically; Romberg's symptom was more marked; he complained of weariness and paræsthesiæ.

In September, a swelling of the right thigh and leg came on suddenly, it was of doughy consistence, and the skin over it was red and hot; the signs of a commencing arthropathy of the left knee-joint were also observed. In March, 1889, he was treated by suspension; being suspended on the first occasion for half a minute, and on the next day for a minute and a half. The suspension gave rise to giddiness, palpitation, and to a feeling of oppression in the chest, of which he had once before complained. Four days later he was seized in the night with difficulty of breathing, and died suddenly. To sum up, the characteristic symptons of tabes present in the case were loss of knee-jerk, sluggish action of pupils, ataxy, impotence, lightning pains, paræsthesiæ, affections of sensation, Romberg's symptom. To these must be added the characteristic tabetic arthropathy with its sudden onset on insignificant provocation, and the sudden swelling of the thigh.

Dr. Kuh gives a minute account of the naked eye and microscopical changes found in the organs. Examination of the right knee-joint showed that the femur, tibia and fibula were thickened and massive. The compact substance of the bone was eburnated, the bony trabeculæ in the spongy substance were thick and hard, and there was no medullary cavity. The borders of the articular cartilages were thickened, and in one place beset with little outgrowths. The latter changes were most evident over the internal condyle and in the neighbourhood of the intercondyloid fossa of the femur.

The spinal cord and brain were very carefully examined. In the cauda equina the tissues generally were excessively vascular, the dura mater thickened, and the soft membranes moderately rich in cells. The vessels showed great changes in their walls, the intima being swollen and infiltrated with round cells, which accumulated in places so as nearly to occlude the lumen; the middle coat was also frequently of abnormal thickness. In the nerves collections of round cells were found between the nerve fibres, many of the latter were clearly degenerated, while again there were many normal ones. Over the whole extent of the cord the pia and arachnoid membranes were much thickened, in many spots they were the seat of a diffuse round-celled infiltration, especially over the lumbar region, and there were a few small gummatous swellings. The vessels of the cord and pia arachnoid showed similar changes to those found in the vessels of the cauda equina. The vessels of the healthy parts of the cord were equally affected, so that the degenerations described below could not be attributed to vascular disease. Anterior roots injected but healthy; posterior roots were altered in the same manner as the nerves of the cauda equina.

Throughout the spinal cord there was found sclerosis of the posterior columns, most advanced in the lumbar region, and in cross sections appearing most intense in the posterior internal root zone (tract of Lissauer) while the columns of Goll were affected in less degree, the sclerosis diminishing in intensity towards the posterior fissure. The changes were symmetrical, and of the nature of a parenchymatous degeneration of nerve fibres.

Meningitis and morbid changes in the blood vessels were present in the pons and medulla; the vessels of the vagus nucleus were intensely injected, and there were a few punctiform hæmorrhages in this situation quite recent and lying close to the nucleus.

In the brain there was general meningitis, most intense over the convexity, but also present at the base; the blood vessels of the brain were generally injected, those of the outer layers of the cortex being extensively diseased in those situations where meningitis was most marked, and there was here also exudation of leucocytes and degeneration of nerve fibres. The changes in the nerves were most marked in the neighbourhood of the diseased vessels. The nerve-cells were for the most part healthy, but showed slight changes in those situations where the disease was most advanced. The left island of Reil was the part most affected, here was found intense meningitis, copious round-celled infiltration of superficial layers of cortex, confined to the vessel walls in the deeper layers, degeneration of nerve fibres, and in part of nerve-cells. The parts of the cortex next most affected were the left frontal convolutions, right island of Reil, and the middle of the right anterior central convolution.

Dr. Kuh observes that the symptoms during life, and the morbid charges found after death admit of no doubt as to the nature of the disease. Besides the spinal degenerations commonly found in tabes there was a meningitis extending over all parts of the cerebro-spinal axis, and in the brain preponderating over the fore part of the convexity, but also present at the base. The nature of the morbid changes which were found throughout the vessels of the central nervous system, and of the meningitis was undoubtedly syphilitic. With regard to the question of the connection between the posterior sclerosis and the meningitis, he considers that they both arose from the same cause, namely, the thorough impregnation of the tissues with the syphilitic poison. From a study of 3471 reported cases of tabes he finds that the proportion of those who had undoubtedly suffered from previous syphilis was 61.8 per cent., a number which for various reasons may be considered as most probably below the real proportion.

The extensive disease of the left insula in the foregoing case is remarkable as during life there were no disturbances of speech; the explanation probably lies in the escape of the deeper layers of the cortex. That no paralysis of any of the cranial nerves occurred which would have enabled a diagnosis of intracranial meningitis to be made is to be attributed to the greater intensity of the meningitis over the convexity of the hemispheres. The morbid changes in the nerve-roots of the cauda equina were presumbably caused by the disease of the vessels. Was the treatment by suspension to be blamed for the sudden death? The author thinks not, but that the sudden termination of the case was due to the invasion of the vital centres by meningitis.

J. MICHELL CLARKE.

Boulay on Pseudo-Bulbar Paralysis (*Gaz. des Hôpitaux*, July, 1891.)

PARALYSIS of the tongue, lips, and velum palati forms a group of symptoms, which may be caused by any lesion, acute or chronic, primary or secondary, of the nuclei of the medulla. But these symptoms are not a certain index of lesion of the medulla, for, under certain circumstances, lesions situate above the medulla may give rise to the symptoms of glosso-labio-laryngeal paralysis, and then we have to deal with pseudo-bulbar paralysis.

History.—Little was known of pseudo-bulbar paralysis before the short paper by Dr. Lépine (1) published in 1877, previous papers by Magnus (2) (1837), and by Jolly (3) (1872), having attracted but little notice. Joffroy (4), however, had noticed that the train of symptoms, characteristic of progressive bulbar paralysis, might be produced by bilateral lesions of the brain, "the lips, tongue and velum palati being thus more or less paralysed, it may be difficult to diagnose between cases of labio-glosso-laryngeal paralysis of cerebral and bulbar origin." Lépine (5), in his paper, gives the records of two new cases followed by autopsy, one by Charcot and Oulmont and the other by Oulmont. In both of these the medulla was intact, but the brain was the seat of bilateral and symmetrical lesions. This writer was led to conclude that the symptoms of labio-glosso-laryngeal paralysis might be caused by lesions of the cerebral hemispheres alone, and of such a diagnosis he gives an example.

Attention being thus drawn to the subject, during the following years about twenty cases were recorded in support of Lépine's conclusion, and the existence of pseudo-bulbar paralysis of cerebral origin appeared to be firmly established.

when in 1887 Oppenheim and Siemerling (5) expressed the opinion that the majority of the cases published under the designation of pseudo-bulbar paralysis did not really deserve that name, but that microscopic examination showed small centres of softening in the medulla and protuberance, which explianed the symptoms observed during life. These authors think that pseudo-bulbar paralysis due to cerebral lesion is extremely rare. Since the publication of Oppenheim and Siemerling's paper the existence of a mixed form of the disorder has been ascertained, the cerebro-bulbar. Of this variety three cases have been put on record by Otto (6). Nevertheless, it is certain that the purely cerebral form is not at all uncommon, which is proved by two cases occurring in the practice of Dr. Déjerine, published, one by Leresche (7) and the other still more recently by Münzer (8), in which all the symptoms of glosso-labio-laryngeal paralysis existed, and yet the most careful research could detect no lesion of the medulla.

Pathogeny; *Etiology*.—In the thirty cases which the author was able to find recorded, twenty-four had been followed by post-mortem examination, and in almost all the lesions were bilateral; frequently they were also symmetrical. In five cases, however, one of the hemispheres was intact [Magnus, Nothnagel (9), Kirchoff (10), Ross (11), Drummond (12)]. To the consideration of these cases the author returns further on.

The lesions may be found in the cortex, in the basal ganglia, or in the white substance; occasionally they are confined to the grey substance of the cortex, or ganglia, but more commonly they affect also the neighbouring white substance. It is rare for the diseased patches to be strictly confined to the cortex, but of this class the cases recorded by Magnus (13), Barlow (14) and Rosenthal (15) are examples. Under these circumstances the pseudo-bulbar symptoms are not well marked or are incomplete. The case recorded by Barlow is the most remarkable, for in it there was present, not only glossoplegia, but also aphasia. In each hemisphere there was a patch of softening of the lower part of the ascending frontal convolution extending to the posterior parts of the second and third frontal convolutions. In three other cases the changes were both superficial and deep [Wernicke (16), Féré (17), Raymond and Artaud (18)]. In Féré's case there was in the right hemisphere a large cortical focus of disease, affecting the inferior half of the ascending parietal convolution, the root of the third frontal, the inferior half of the gyrus angularis, and a portion of the first temporal and of the insula; the softening also extended to the external segments of the lenticular nuclei. In the cases of Wernicke and Raymond the lesions of the centrum ovale were predominant.

The basal ganglia and the surrounding white substance are practically the common seat of the lesions, which may be extensive and diffuse, occupying simultaneously the two ganglia of the corpus striatum, the optic thalamus and the surrounding white fibres [Ochs (19), Drummond, and Berger (20)], but occasionally the lesions are circumscribed, and are on that account all the more interesting. Under such circumstances, the white substance being but slightly affected, the lesions involve chiefly the basal ganglia, the lenticular nucleus, and especially its external segment (putamen), being chiefly attacked, to the exclusion of the optic thalamus and caudate nucleus. Such was the case in the patients of Oulmont, Ross, Füller, and Browning (21), where the lesions were localised in the extra-ventricular nucleus of the corpus striatum, and but slightly involved the white substance. In the cases recorded by Auscher (22) and Lereschc, in which the pseudo-bulbar symptoms were more clearly defined, the lesions were exactly confined to the putamen. In one case only, the lesions, though confined to the basal ganglia, affected the optic thalamus and caudate nucleus, leaving intact the lenticular nucleus, [Eisenlohr (23)], but here the glosso-labial symptoms were not complete. The lesions are, as a rule, symmetrical, but this condition is not invariable; in Hahn's case, for instance, there were two foci of softening, one in the right caudate nucleus, the other in the white substance of the middle lobe of the left hemisphere. In addition to these lesions of the cerebrum there have been found occasionally foci in the cerebellum (Auscher) or in the protuberance (Berger), but in none of the cases referred to has there been any lesion of the medulla.

Thus it is seen that except in some unusual cases in which the foci of disease were limited to the cortex, the most constant lesion is one confined to the lenticular nuclei, and especially to the external segment.

The lesions most common are foci of softening, or of hæmorrhage, presenting various characteristics according to their age. In one solitary case (Jolly) there was sclerosis of the white substance of the hemispheres, with several patches of sclerosis in the anterior portions of the peduncles, the integrity of the medulla being proved by microscopic examination. Gummata of the meninges or brain, as shown by Münzer's case, may also give rise to the group of symptoms characteristic of glosso-labial paralysis. In addition to two gummata of about the size of a pea on the convexity of each hemisphere, there were, in that case, gummatous patches in the right putamen and in the head of the left caudate nucleus.

Histological examination of the paralysed muscles has given no result, Berger, Ochs and Leresche having in vain sought for traces of atrophy.

After the lesions of the brain the change most commonly met with is atheromatous degeneration of the arteries, and especially of the arteries of the base. In this disease is seen the most common cause of the multiple foci met with in the brain, atheroma having been noticed in more than half the recorded cases, and occasionally it was of so pronounced a character as to seem almost capable of causing compression of the medulla, but in the absence of bulbar lesion the author does not believe that the symptoms observed during life could have been thus produced.

The lesions of pseudo-bulbar paralysis being, as a rule, the result of atheroma, it will be readily understood that the disease is especially one of advanced life. In fourteen cases, that is in more than half of those reported, the patients were over sixty years of age. When the symptoms appeared in children, or in middle life, the cerebral foci were due to embolism. This was found in Barlow's case, in which a child of ten, the subject of aortic disease, as a sequel of embolisms in the two Sylvian fissures, suffered from double facial paralysis, with paralysis of the tongue, and difficulty in swallowing. Kirchoff's case is similar to the above; a youth of twenty-four was attacked by cerebral embolism caused by mitral disease. Syphilis in the young might also be a cause, not only by giving rise to gummata, but also by syphilitic disease of the arteries. No case, however, has been reported absolutely proving this assertion, though in one of Berger's patients, beyond doubt the subject of syphilis, arteritis was found in the two Sylvian fissures, but it is not mentioned that this arteritis was of syphilitic origin. In a case recorded by Professor Fournier (25), paralysis of the lips, tongue and velum palati was developed in a patient suffering from cerebral syphilis with aphasia and repeated hemiplegia, in whom recovery took place under anti-syphilitic treatment.

Symptoms.—The facial expression of one suffering from this disorder is of a striking character; the upper part of his countenance is mobile, while the lower part is flaccid and sluggish. The eyes are well opened and the forehead wrinkles readily, while on the other hand the naso-labial grooves are ill-defined, the cheeks are flaccid, and the half-closed lips allow the saliva to trickle from their angles. The patient wears an expression at once of astonishment and anxiety. This dull, stupid expression is increased when the patient is made to talk. He understands what is said to him, but his answers are comprehended with difficulty, the sounds to which he gives utterance being almost unintelligible. He does not hesitate about his words, which come to him without mental effort, but his articulation is embarrassed and monotonous. His lips hardly open to allow the words to escape, and the words are so badly pronounced that one can but catch a few syllables here and there. The patient understands what he reads, and reads aloud without correcting himself, but always with a stammer. It is seen that the tone of the voice is not normal; it is more or less nasal. The linguals and the vowels "o" "u" are especially indistinct. Occasionally the voice is enfeebled, and there is a certain degree of aphonia.

The patient is not aphasic; his remembrance of words is intact; he speaks, reads, writes, without mental effort. He has that particular difficulty of articulation which has been described under the names dysarthria and anarthria, a difficulty pointing to a paralysis of the lips, tongue and velum palati, and perhaps of certain muscles of the larynx.

Occasionally the paralysis of the lips is not very well marked, and we have to deal rather with a paresis than with a paralysis. It often happens that the two sides of the face are not equally affected, and then there is a more or less pronounced want of symmetry of the mouth, and on one side the cheek is less flaccid, and the naso-labial furrow better marked than on the other. The zygomatici and the levatores anguli oris, together with the muscles of the chin, are more or less affected, and this produces habitually its functional consequences.

The movements of the tongue are always embarrassed, but never completely lost. This organ moves slowly and with difficulty; the tip is not readily raised towards the roof of the mouth, but it is the lateral movements which are especially affected. The patient is unable to move the tip of his tongue between his gums and cheek, and, though still able to protrude it, he does so with slight deviation towards the more paralysed side. The dorsum of the tongue can no longer be furrowed so as to form a groove, and when the paralysis is still further advanced the tongue lies flaccid on the floor of the mouth.

The velum palati is not, as a rule, equally paralysed on the two sides, but presents a slight want of symmetry; one of the pillars is lower than the other, or rather the uvula is slightly deviated. When the patient phonates the pillars approximate incompletely. Sometimes the velum is absolutely flaccid and inert.

This association of various paralyses causes not only difficulty in articulation, as in Duchenne's disease, but also gives rise to difficulty in mastication. Deglutition is considerably embarrassed, solids being swallowed easier than liquids, which are at times ejected by the nose. The patient drinks a little at a time in small sips, throwing his head back as he does so. Occasionally he has a fit of coughing, which shows that a few drops of the liquid have reached the larynx. The saliva flows almost continually from one of the angles of the mouth, especially when the patient is in the erect position ; in the horizontal position it is swallowed with less difficulty, but as soon as he is erect, or inclines his head forward, the saliva runs away drop by drop. Mastication is difficult, the hollows between the teeth and cheeks remaining full of alimentary particles; food falls occasionally from the mouth, and when the paralysis is in an advanced stage the sufferer is seen to aid deglutition by thrusting the food into the pharynx with his fingers. These difficulties attain their maximum when the muscles of the lower jaw become affected. Paralysis of these muscles has been observed in several cases. In Charcot's case it is said that the jaws could not be separated more than two centimetres at the utmost. Oulmont's patient opened his mouth with difficulty, and in order to do so completely the hand had to be used to depress the lower jaw. In Barlow's case mastication was impossible, and Browning's patient was unable to open his mouth. Magnus and Feré noticed, in their patients, the weakness and slight extent of the movements of the lower jaw. Sometimes the mouth remains half open, and the patient cannot bring his teeth together without an effort, while in other cases the depression of the lower jaw is difficult. When the pterygoid muscles are weakened the lateral movements also become embarrassed. In opposition to what occurs in true progressive bulbar paralysis, this paralysis of the muscles of mastication does not mark a fresh stage in the progress of the malady, but when it is found it is at the very commencement of the illness.

The last remark may be applied to the laryngeal trouble which has been noticed in patients suffering from pseudobulbar paralysis, but there is some doubt as to its occurence, owing to the infrequency of laryngoscopic examination in such cases. Oppenheim and Siemerling deny its occurrence. In the patient of Füller and Browning the voice was lost and replaced by a sort of guttural sound. In other cases the patient can but utter a few feeble inarticulate sounds. In one of Professor Charcot's patients the aphonia was almost absolute, and it was only by a violent effort that he succeeded in giving utterance to an indescribable grunting noise. Auscher's patient could only speak with stifled voice. Unfortunately, laryngoscopic examination has been but seldom made in these cases. In two patients, however, in whom Professor Charcot had diagnosed pseudo-bulbar paralysis of cerebral origin, Cartaz (26) found difficulty in the adduction of the vocal cords, due to paresis of the thyro-arytenoid, and paralysis of the arytenoid muscles. The result of the autopsies on these patients is unknown to the author. In Münzer's case the vocal cords did not approximate either in phonation or deglutition, and microscopic examination after death demonstrated the absence of any lesion of the medulla. From the above it would appear that in pseudo-bulbar paralysis, we meet with laryngeal disorder characterised by paralysis of the muscles of phonation. Hitherto the abductors of the vocal cords have been found intact and respiratory difficulty of laryngeal origin has not been observed, beyond what has been stated in the foregoing remarks.

Such is the common distribution of the paralysis in a typical case of labio-glosso-laryngeal disease. But in every case the symptoms are not all present; and, besides the cases where the lips, tongue, velum palati, and muscles of phonation are all involved, some are met with where one or other of these organs has escaped. Thus in one of Ross's patients the velum palati was still moveable, while movement of the lips and tongue was no longer possible. It even happens occasionally that the paralysis affects but one organ; the tongue (Rosenthal), or the lips (Eisenlohr) being alone affected.

One characteristic common to all these forms of paralysis, be their distribution what it may, is the absence of atrophy of the affected muscles. The tongue retains its usual volume and consistence, and its surface remains smooth; the lips, also, preserve their normal thickness and there is, as a rule, no fibrillar tremor.

The condition of the reflexes varies. Frequently the pharyngeal reflex is diminished, but slight contraction of the velum palati being provoked by stimulation of the pharynx; and prolonged stimulation being required to provoke cough and nausea. Sometimes the finger may be swept round the fauces without causing any reaction; but, on the other hand, there may be an exaggeration of the masseteric reflex, for on abruptly depressing the jaw, a species of epileptoid tremor is produced.

In opposition to what is seen in true bulbar paralysis, the electric reaction of the paralysed muscles is normal; there is neither the reaction of degeneration, nor even any diminution of excitability to the faradic current.

The tactile sensibility of the mucous membrane of the lips, tongue, and pharynx is preserved; even when the pharyngeal reflex is slow and even abolished, the patient continues to feel the slightest touch. Special sensation is as a rule intact, instances of sensorial trouble having been but rarely noticed.

In addition to these paralyses of the muscles of the face and neck, paralysis of the limbs is found in most of the cases. Nevertheless, the patient can generally walk, sometimes with the gait commonly seen in hemiplegia, but more frequently, and that especially when he has suffered from repeated attacks of paralysis of the two sides of the body, the gait assumes the special character to which Déjerine has drawn attention. This was recently observed by the author in a patient of Professor Debove; who while suffering from arterio-sclerosis, was seized by acute difficulty of speech, with paresis of the lips and tongue, the retrospective diagnosis of pseudo-bulbar paralysis being justified by *post-mortem* examination. In each hemisphere there was an old patch of softening in the external half of the lenticular nucleus, and external capsule. In spite of the previous absence of paralysis, walking had by slight degrees become difficult:

the patient progressed by short steps with his body slightly inclined forward, hardly raising his feet from the floor, and getting over the ground slowly, although his steps were taken with relative frequency. The same characters of the gait are noticed in several of the cases. "When standing there is something in the patient's carriage suggestive of paralysis agitans; he does not hold himself erect, his body being slightly inclined forward with the thighs and legs slightly bent. When he is examined in the recumbent position, it is seen that all the movements are preserved, but that they are executed slowly and with slight limitation in their extent" (Leresche). In the upper limbs this difficulty of movement is less marked; it has been noticed, however, that the sufferers have a certain difficulty in dressing and undressing themselves.

The mental abnormalities occasionally observed in patients suffering from pseudo-bulbar paralysis present no special characteristics, but closely resemble those met with in hemiplegia. Loss of memory, mental slowness, incoherence of ideas and even dementia have been observed. The patient laughs without sufficient cause; frequently he is irritable, and now and then, outbreaks of anger, alternate with laughter or tears. In connection with these bursts of tears a curious phenomenon has been noticed by several observers: the patient while unable to laugh voluntarily, still does so involuntarily in these outbreaks. Here then is a true antagonism between the voluntary and involuntary movements of the face (Lepine).

Progress, Duration, Termination.—Generally the symptoms indicative of glosso-labial paralysis come on suddenly in an apoplectic seizure in a patient who has on previous occasions suffered from apoplexy, either with, or without transitory aphasia. The hemiplegia, as a rule, improves more or less, and sometimes even passes away completely, leaving great weakness of the limbs. The pseudo-bulbar symptoms alone persist with alternations of improvement or increase. In other cases pseudo-bulbar paralysis makes its first appearance in an attack of apoplexy in a patient who has not previously suffered from hemiplegia. In rare cases it appears without paralysis, or loss of consciousness: after an ordinary attack of giddiness the patient is found to have paralysis of the lips and tongue (Hahn, Kirchoff, Ross, Raymond and Artaud), the symptoms thus initiated, remaining perhaps the only morbid phenomena up to the death of the patient, unless he eventually experiences an attack of hemiplegia so common in the histories of these cases. The symptoms may remain stationary, or may for a time improve, or may increase in severity. This aggravation of the symptoms is sometimes spontaneous but more frequently it is the consequence of a fresh attack of apoplexy, and is then sudden in its onset. The evolution of the affection is neither regular nor progressive. The respiratory and cardiac symptoms, the attacks of dyspnœa and syncope, which so frequently terminate cases of progressive bulbar paralysis, are not met with in pseudo-bulbar paralysis, or, to be more correct, they are quite the exception. Münzer's patient alone among the recorded cases died from cardiac and respiratory troubles, which might have been attributed to changes in the nuclei of the pneumogastric nerves, had not their integrity been demonstrated by histological examination.

The duration of the lives of patients suffering from pseudo-bulbar paralysis is extremely variable. Some, and these form the majority, die a few months after the onset of the disease; others are carried off more rapidly, and survive but a few days. In other cases again the lesions are more circumscribed, and unless a fresh attack takes place, the patient survives several years: one patient did not die until eleven years after the onset, when his death was caused by a broken leg (Berger).

A fresh apoplectic attack is the most common cause of death, which however, as in Duchenne's case may be due to difficulty in deglutition. Particles of food may find their way into the respiratory passages, and set up, if not asphyxia, at any rate a fatal broncho-pneumonia. Now and then it is found necessary to feed the patient with the stomach-pump and he dies gradually from marasmus and cachexia. Occasionally albuminuria, from which these patients frequently suffer, increases the cachexia, or causes fatal complications.

Recovery appears possible when the cerebral lesions are caused by syphilis, and are recent and not destructive. The improvement observed in some cases of pseudo-bulbar paralysis is almost always of a transitory character.

Prognosis is worse where deglutition is affected, or when the state of the heart or vessels gives rise to an expectation of fresh attacks of apoplexy.

Diagnosis.—This is frequently difficult, and always requires care. We may first eliminate the danger of confusion between this affection and double facial paralysis of peripheral origin, and diphtheritic paralysis of the velum palati; the former is characterised by the total paralysis of the facial nerves, with integrity of the tongue; the latter, while attacking the velum palati, leaves intact the tongue and the lips.

In most cases we are in one of the two following positions: either, having an incomplete knowledge of the onset of the illness, we mistake it for some chronic bulbar affection; or knowing that the labio-glosso-laryngeal symptoms have appeared suddenly in an apoplectic attack, we erroneously fancy that we have to deal with an acute disease of the medulla, such as bulbar apoplexy.

In spite of the similarity between pseudo-bulbar paralysis of cerebral origin and Duchenne's disease, there are, nevertheless, differences between the two easily seen when they are compared. The author gives a *resumé* of the symptoms of these two affections in order to bring into relief the differences between them:

Pseudo-bulbar paralysis.—Onset generally sudden and during an apoplectic attack. Simultaneous paralysis of the tongue, lips, and velum palati, generally reaching its maximum at the first onset, but sometimes aggravated by a fresh attack. This paralysis is frequently more marked on one side. No atrophy of the paralysed muscles. Reflexes not affected, at any rate at the onset. Electrical reactions normal. Mental condition similar to that characteristic of cerebral softening (loss of memory, mental weakness), repeated apoplectic attacks, paralysis of the limbs.

Progressive bulbar paralysis.—Onset gradual, without obvious cause. The tongue, velum palati, lips, and later on the muscles of mastication, and the larynx attacked one by one. Their paralysis increase progressively in intensity; they are symmetrical and cause no deviation of the tongue, uvula, or angles of the mouth. The muscles are the seat of fibrillar tremors, and undergo a more or less marked degree of atrophy. Reaction of degeneration generally present. The intelligence unaffected to the close of the affection; no paralysis of the limbs. The laryngeal paralysis, dyspnœa, and fainting fits, though more frequent in progressive bulbar paralysis, have not an absolute diagnostic value, having been observed in several alleged cases of pseudo-bulbar paralysis.

Should the symptoms of glosso-labial paralysis appear suddenly during an illness, with or without paralysis, the hypothesis of a bulbar lesion can not, at once, be rejected. There is, in fact, a whole group of acute bulbar affections, capable of producing, with more or less fidelity, the symptoms of glosso-labial paralysis, such as emboli and hæmorrhages of the medulla, acute myelitis, and in certain cases compression of the medulla.

Apoplexy of the medulla, when the patient survives the onset, is generally accompanied by difficulty of articulation and of deglutition similar to that of pseudo-bulbar paralysis; the paralysis of the lips, tongue and velum palati may even be the only symptoms, and the diagnosis is then almost impossible. But in most cases other symptoms such as alternate hemiplegia, paralysis of both halves of the body coming on simultaneously, respiratory and cardiac symptoms, vomiting and urinary troubles, will point to the medulla as the seat of the lesion. These phenomena frequently appear without loss of consciousness, and unaccompanied by any mental disturbance. Eventually disease of the medullary nuclei causes atrophy of the paralysed muscles.

The author does not think it necessary to do more than mention acute myelitis of the medulla, of which but three cases have been recorded; the clinical history of this affection presents a close resemblance to that of embolism of the medulla.

Tumours, especially aneurisms of the basilar or vertebral arteries and syphilitic gummata in the neighbourhood of the medulla have, under exceptional circumstances, given rise to all the symptoms of medullary paralysis, such as difficulty in swallowing, dysarthria, and difficulty in respiration. The absence of cerebral symptoms and the simultaneous paralysis of all four extremities will, as has been pointed out above, make clear the diagnosis.

Finally the diagnosis between pseudo-bulbar paralysis of sudden onset and acute medullary paralysis is only possible in as far as certain symptoms, such as aphasia, hemiopia, &c., and mental abnormalities demonstrate the preponderant participation of the brain.

As to the clinical distinction which Oppenheim and Siemerling have attempted to establish between the pure form of pseudo-bulbar paralysis and the impure or cerebro-bulbar, the author thinks it incapable of being maintained.

The appearance, from lesion of the brain, of a group of symptoms generally associated with disease of the medulla may, in some degree, be explained by the aid of modern researches in anatomy and physiology. It is now known that the medullary nuclei are not absolutely autonomous centres; each has a corresponding centre in the brain, from which, by means of uniting fibres, it receives its impulse. It would appear that any lesions of these centres or cerebro-medullary fibres would cause embarrassment of the corresponding centres in the medulla, though the structure of the latter remained intact. The distribution of these lesions may be of such a character as to paralyse simultaneously the lips, the tongue, and the velum palati. That these organs should be paralysed by lesion of the brain is not a matter of hypothesis, but is seen daily at the bedside.

In an ordinary attack of hemiplegia due to lesion of the internal capsule, amongst other symptoms, we find paralysis of the lower part of the face and of one side of the tongue, together with some difficulty in swallowing, and deviation of the uvula. That is to say there is in common hemiplegia a certain degree of unilateral glosso-labial paralysis. If in such a case an identical lesion of the opposite hemisphere were to be produced, we should have paralysis of the two lower halves of the face, two halves of the tongue, and velum palati, a group of symptoms analogous to what is seen in bulbar paralysis.

Thus it is evident that bulbar paralysis is closely simulated when the centres in the brain corresponding to those in the medulla, or the white commissures, are attacked by symmetrical lesions. But, unfortunately, little is known of the distribution in the brain in the centres homonymous to those in the medulla, the very existence of some of them being quite uncertain. As to the commissures their course is to a great extent hypothetical. Some of these centres may, however, be localised with a certain degree of precision. The centre for the movements of the lips (inferior facial) is situated in the lower part of the ascending frontal convolution, and close to the latter, possibly identical with it, are the centres for the movements of the tongue (hypoglossal) and for the muscles of mastication (motor branch of the fifth). According to the experiments of Krause, and of Horsley and Schäfer, the centre for deglutition is also in the lower portion of the ascending frontal convolution. The existence of a cortical centre for the larynx, though still somewhat uncertain, is becoming day by day more probable. Semon and Horsley, from physiological experiments on monkeys, believe it to be placed at the point of union of the lower part of the ascending frontal with the third frontal convolution; and this is the place also suggested by many clinical and pathological observations.

These centres, it will thus be seen, are all in the cortex, and are grouped together in so small a space that the centre for the lips is closer to the hypoglossal centre than to that for the upper portion of the face. They are, moreover, bilateral, but it must be noticed that unilateral excitement of several of them sets up bilateral movements of the muscles they supply, and, on the other hand, that unilateral destruction does not cause paralysis. These centres thus seem to be able mutually to replace each other. But when a bilateral lesion takes place a corresponding paralysis of the organs occurs. This theory, though applicable to cortical or sub-cortical lesions, is most unfortunately at fault when we have to deal with cases in which the changes are limited to what is their most common seat, the lenticular nucleus. Two hypotheses have been put forth to explain the symptoms following lesions of the extra-ventricular nucleus.

(1.) That this nucleus acts as a special centre regulating the movements of speech, &c.

(2) That the symptoms observed are due to the destruction of white fibres, which arising from cortical centres, pass through the lenticular nucleus in order to reach the peduncle. The physiology of the intra-cerebral ganglia being almost unknown, the first hypothesis rests upon a vague anatomical basis; the histological identity of the corpus striatum with the grey substance of the cortex, and the existence of nerve fibres (intermediate band of Meynert), which, springing from the putamen, pass to the peduncle, then to the protuberance and perhaps to the medulla.

The second hypothesis has a greater appearance of truth, as it allows us to give to pseudo-bulbar paralysis due to lesions of the lenticular nuclei, the same interpretation as when the cause is cortical or sub-cortical. It is known that the fibres arising from the cortex do not all converge towards the internal capsule, but that certain of them springing from the superior and external surfaces of the hemispheres make a detour in order to reach the peduncles. After traversing the different segments of the lenticular nucleus these fibres contribute to form the ansa lenticularis, and then join the bundles of fibres coming from the internal capsule. The origin of all these fibres is not known with certainty, but it has been shown that a certain number of them come from the upper facial cortical centre, and by analogy it may be supposed that some of them arise from other cortical centres, especially from those the injury to which causes the group of symptoms called glosso-labial paralysis. The destruction of these fibres, will on the above supposition, explain the symptoms seen to follow bilateral lesions of the lenticular nuclei.

If the truth of the following remarks, borrowed from

Kirchoff, be granted, the same explanation may be given of the five or six cases in which the lesions were unilateral. On this supposition the symmetrical muscles, which, as a rule, act simultaneously, (tongue, larynx, &c.) will be represented by centres in each of the two hemispheres, but normally only the crossed fibres will be in use. When one of the hemispheres is injured, the other may supply its place, and complete conduction takes place. But if one of the hemispheres, by some anomaly of its structure, or by an acquired habit (as takes place with language), is alone able to act, then its lesion will, of necessity, bring about loss of function.

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Richard Atkinson, F.R.C.S.Eng.

Syringomyelia and Morvan's Disease.

I.—*Paresis and Analgesia of the Upper Extremities associated with Whitlows. Paresi-analgesia*[1] *of the Upper Extremities or Morvan's Disease.* A review by F. Verchère (*Revue des Sciences Médicales*).

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[1] Paréso-analgésie of French writers. It has been thought desirable to alter the form of the word in English.

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Morvan's whitlow was first described in 1883. It attracted a great deal of attention and was much discussed by surgeons, to whose province it was thought to belong. All agreed as to its importance, and its discovery was hailed as that of an entirely new and distinct disease.

It was not till 1888 that the current of opinion was turned into a new channel. This was the result of a decisive autopsy in which the typical features of Morvan's disease were found to be associated with the lesions of syringomyelia. From that date Morvan's whitlow passed from the sphere of surgery to that of medicine. It was impossible to resist the conclusion based as yet on a single, but crucial, observation. Further evidence, had any been needed, was quickly forthcoming to settle on a secure basis its true position in nosology. It may now perhaps be asserted that this has been done. Morvan's discovery, deprived of its independent consequence, has gone to swell the significance of a train of symptoms concerning which our knowledge is steadily growing, and which already surpasses in interest and complexity the earlier acquisitions of neurology. In thus deciding, however, there is danger that we prejudge the question. Besides that, M. Morvan himself resolutely rejects the new pathology, there has been in another quarter a determined effort to reopen the discussion. It had long been recognised that certain forms of syringomyelia, and amongst them those that conformed most closely to the type of Morvan's disease, bore a strong resemblance to anæsthetic leprosy. A difficulty originating in this source was foreseen rather than experienced when Professor Charcot undertook to lay down the principles of a differential diagnosis. The immediate result was a fresh controversy, in which it was maintained by the new disputants that the grounds of such a diagnosis were insufficient, and that, in fact, there were adequate reasons to postulate the identity of the two diseases. *Adhoc sub judice lis est*, and the event may prove that Morvan's disease has not yet seen its final vicissitude, and that it is destined to be handed over to the dermatologist, who claims it as a variety of a contagious parasitic disease.

Under these circumstances it behoves the reviewer to maintain an impartial attitude; to abstain from undue criticism and content himself with noticing the more important contributions and stating the arguments adduced for the successive transfers of this remarkable malady from one department of medical science to another.

It will be seen from what we have said that the history of the disease is divided into three periods, corresponding to three different phases of opinion, and the literature of the subject is similarly marked out. The dates are determined in each case by the suggestive research or the epoch-making treatise, which has given a new turn to thought or shattered former convictions.

The three phases or periods may be arranged as follows :

1.—*Period of clinical surgery.* Morvan's whitlow. A morbid entity having an external cause. Account of the characteristic symptoms of such morbid entity, 1883-1888.

II.—*Period of pathological anatomy.* A medical incident. Association with syringomyelia. Account of syringomyelia (Type Morvan), 1888-91.

III.—*Dermatological period.* Anæsthetic leprosy and syringomyelia, 1891.

First Period.—Clinical. A surgical disease. Morbid entity having an external cause. Account of the symptoms belonging to such morbid entity, 1883-1888.

The first period begins with the original description of the disease. In 1883 Dr. Morvan, of Lannilis, published in the *Gazette Hebdomadaire* his observations in seven cases of an affection which he defined as "paresis and analgesia of the upper extremities, associated with whitlow," otherwise "paresi-analgesia of the upper extremities." These observations are a model of accuracy and acumen. It is but just to record that amongst the voluminous contributions to the subject which have since appeared. not one has had to correct or to amend a single statement of those made by Dr. Morvan in the first instance. It was inevitable that ampler experience should find some details to add, and the event has proved that a place in the family of diseases, not then foreseen, awaited the new discovery; but from first to last Morvan's description remains the unembellished counterpart of the symptoms to which he gave the name of paresi-analgesia. These symptoms are summed up in three words—paresis, analgesia, and whitlow. They are the same now as they were at first. Taking each of these symptoms separately, we shall enquire what are their characters and in what way they are subject to modification in different patients.

Analgesia extends to all the modes of sensation, but it varies in its distribution in a manner often corresponding to the stage of advancement which the disease has reached. Where there has been only one whitlow the analgesia, as a rule, is limited; but where, on the other hand, these have successively formed during a period, perhaps, of many years, and when the skin is thickened and chapped there is apt to be complete analgesia, involving the whole of one or both upper extremities; more rarely the lower limbs and the face and trunk are similarly affected. Even in such cases, however, the condition is not absolute, some portions of the surface within the affected area retaining their sensibility to pain. And it is to be observed that the patches in which sensibility persists are not disseminated.

Analgesia proceeds in a uniform manner and implicates entire segments together. It is most extensive on the side first attacked, the hand, fore-arm and arm, perhaps, being absolutely involved; while, on the other side, a more limited area, as the hand and fore-arm only may be affected.

To analgesia are frequently added other derangements of sensation: insensibility to touch or to temperature may be also present. The dissociation of the different modes of sensation are common, but not invariable. Its occasional manifestation is a fact of great importance, and it will be seen that it has been made the basis of remarkable inferences by writers in the second period of the history of paresi-analgesia. Neuralgic crises of the affected limb are also observed. These crises take place at first at uncertain intervals, and finally cease when the disease is fully established. Morvan, writing in 1883, says, "Paralysis of muscles in the fore-arm and hand is constantly present, and this is accompanied by atrophy. Paralysis and atrophy are of variable extent. They may occupy the entire limb, but as a rule they are confined to the part below the elbow." Subsequent observations have not confirmed this statement. Paresis was absent in the cases reported by Guilliot and Broca, and in those which came under Morvan's own notice at a later date (1886). Paralysis, when it occurs, is not limited to voluntary movement, but may also be demonstrated by electrical excitation. Morvan observed that the muscles responded badly to faradic currents. Neither, however, is this invariable, and Hanot and Ogier de Spéville have found faradic contractility unimpaired. The reflexes are subject to great variety. The olecranon and patellar reactions are sometimes abolished; in other cases retained or exaggerated. The formation of whitlows is the essential characteristic of Morvan's disease. It is occasionally the earliest lesion. Sometimes it is preceded by neuralgic pains, with a sensation of weight in the threatened limb, and it may be that analgesia has previously existed for a considerable time, so that the patient inadvertently burns himself before it is found out. In many cases, however, whitlow is the first symptom. It has all the appearances of the ordinary inflammation, but without the pain, and when it has been decided to make an incision the operation is borne with complete indifference. There is no pain complained of, and the part heals readily as a rule, but usually with the loss of a phalanx. In fact, the bone is in most cases necrosed, though the process is slow and painless, with nothing to mark its character. No sooner is the first whitlow removed and the finger healed than another begins to form, and this in its turn is followed by many more. The different fingers are successively attacked, each after an interval of uncertain length. The sufferers witness their hands becoming progressively deformed, their fingers, so to speak, dropping off one after another. The unfortunate patient, who is without independent means, "is rendered unable to work; compelled at first to supply his hunger by the means of some precarious occupation, he is driven sooner or later to seek support at the public expense" (Charcot).

The upper limbs are not alone subject to mutilation. The same process may fix upon the feet and produce a similar deformity of the toes. Perforating lesions take place, and life may be prolonged for many years, while the patient is incapacitated and under the constant apprehension of renewed attacks.

Such is a broad outline of the characteristic symptoms of Morvan's disease. This description has been taken for the most part from an excellent work by M. Ogier de Spéville, which in a sense marks the climax in the history of that affection.

It is necessary, however, to bestow some attention on points which have been elicited by subsequent observations. Amongst these are sensory derangements, such as impairment of sight and of hearing, loss of taste and smell, and affections of sensation involving the head and face. A joint affection of the character of a dry arthritis may take place. This was first shown by Prouff, and afterwards confirmed in his cases by Morvan. The latter has also observed a double fracture in the lower third of the fore-arm, caused by moderate exertion.

Finally, Auguste Broca and Morvan have directed attention to the frequent occurrence of scoliosis in connection with paresi-algesia. Of twelve patients six furnished instances of the deformity. Its significance has been much discussed. The main question is as to whether scoliosis should be regarded as the cause or as the consequence of the nerve lesions. The monographs of Morvan, Prouff, Broca, and a chapter in de Spéville's work are devoted to the controversy. Morvan, quoted by de Spéville, maintains (1) that Broca and Prouff were misled in supposing that curvature of the spine is antecedent to paresi-analgesia ; (2) other affections of the central nervous organs are not accompanied by scoliosis, and this is a circumstance of considerable weight. "As to the first of these statements," observes Ogier de Spéville, "the matter in dispute is of little consequence, and we have no hesitation in hazarding the opinion that the cases from which Morvan, on the one hand, and Broca and Prouff, on the other, draw their conclusions, were not comparable. In the latter, where the subjects were young persons of thirteen or fourteen years and where the curvature had its convexity to the right in the upper part of the dorsal region, it was undoubtedly of the usual adolescent character. Here it seems impossible that syringo-myelia could have preceded the deformity. On the other hand the order favoured by Morvan is much the more probable, in view of the facts reported by him."

With reference to the second of Morvan's positions, that in which he attributes a secondary spinal curvature to paresi-analgesia alone of nervous disorders, it is not in accordance with facts. Pitres (of Bordeaux) and Kœnig have established its existence as a result of locomotor ataxy, and Pierre Marie has met with remarkable instances in the disease, to which he has given the name of acromegaly.

We have dwelt at some length upon this point because the spinal deformity has played a conspicuous part in the history of Morvan's disease. To complete the tale of symptoms it remains to mention that besides the chaps and fissures of the skin covering the extremities there is a liability to deep ulceration, which may give rise to abscess in the sheaths of the tendons, and which has been compared to perforating ulcer of the palm. The hands may be the seat of a pustular eruption, their temperature is often considerably lowered; they assume a livid purple colour, and with the fingers deprived of their nails, mutilated and deformed, the whole member presents a grotesque and pitiable appearance. The disease runs a long and irregular course, and may be in progress for ten, fifteen, or twenty years. In Prouff's case there was an interval of twenty years between two consecutive whitlows.

At this period the pathology of the disease was a source of perplexity. There was little to be learnt from its apparent causation. Though it occurred most frequently after forty, it might appear at any age from twelve to sixty. Males were attacked more frequently than females in the proportion of thirty to twenty (Ogier de Spéville). This may be accounted for by the fact that the starting point of the original lesions were attributable to external causes, injury or frost-bite (Morvan, Czerny) though it was also traceable to a previous illness, such as smallpox, measles, malarial and typhoid fevers. Finally, in the case of a dyer whose hands were habitually immersed in irritating fluids, localised toxic symptoms were observed.

A knowledge of these circumstances was calculated to throw little light on the matter. In addition there was the result of a single autopsy conducted by Gombault and Reboul on the subject of Prouff's case. These observers found extensive changes in the peripheral nerves. The fibres were destitute of myelin, and they further exhibited a considerable thickening of the areolar investments and endoneurium—in short, all the evidence of a pronounced neuritis affecting both the nerve structure proper and its interstitial elements. They therefore determined in favour of a peripheral origin, and came to the conclusion that the essence of the disease was a trophic disturbance associated with neuritis. There was still room to speculate as to its causation. A theory was suggested by the comparative frequency with which cases were met with in the district where Morvan and Prouff practised, and the disease was attributed to endemic influence. This hypothesis was revived in 1890 by Déjerine and Tuilant. "The affection," they said, "is undoubtedly local in its incidence, and the proof is that M. Morvan has encountered twenty cases within the two cantons where he practises. Pending more certain information the analgesic whitlow must be regarded as a peripheral neuritis of a toxic or infectious character, and depending upon a cause as yet undiscovered." Gombault and Reboul had previously entertained the notion that the neuritis might have its cause in a specific contagion derived from fish. In this way the ground was prepared for an association of Morvan's disease with leprosy.

This hypothesis of a peripheral neuritis, however, having held the field for a brief space was destined to be again disposed of. Thibierge, in his remarkable work on disorders of the skin in syringomyelia, observes that the importance of peripheral nerve changes needs to be discounted. They are met with in so many and such different connections that their pathological value is open to question. Gombault's communication to the Société Anatomique (July 25th, 1890), Brissaud's to the Société de Biologie (July 25th), and a valuable critique by Babinski (*Gaz. Hebdom.*, 1890, pp. 374, 383), go to prove how guarded should be the interpretation of peripheral lesions. These considerations were fatal to the provisional pathology of Morvan's disease.

It was reserved for new discoveries to carry conviction and to remove ill-founded prejudices. The revolution was not long pending, and in 1887 it was accomplished. From that year dates the second epoch in the history of paresi-analgesia.

Second Period.—Period of pathological anatomy. Morvan's whitlow a medical incident. Association with syringomyelia. Account of syringomyelia (Type Morvan), 1888-1891.

The first attacks upon the individuality of Morvan's disease were the result of pathological investigation. Reference has been made to the observations of Monod and Reboul, and the consequent attempts to account for the symptoms by changes in the peripheral nerves, such as were to be seen in the digital branches of the amputated fingers. Soon after similar lesions were observed by Gombault and Reboul in the autopsy which they performed on Prouff's case, with the addition, however, of other changes in the spinal cord. Unfortunately the existence of extreme spinal curvature had interfered with the success of the examination by rendering the extraction of the cord very difficult. The changes which it exhibited were confined almost entirely to the cervical enlargement. They consisted of an increase in the connective tissue of the posterior columns and posterior horns of the grey matter, with thickening of the vascular walls. Similar appearances were to be detected in the lumbar region. "The central lesions were, on the whole, much less considerable than those of the peripheral nerves." The latter were restricted to the upper extremities. Gombault and Reboul are not disposed to admit that the loss of substance in this case corresponds to the cavities of syringomyelia, but "there can be no doubt that the lacunæ bear a remarkable resemblance to those which are often seen in the cavities in question; neither is it unreasonable to suppose that the walls of such a cavity may give way under pressure, and subsequently come together with the approximation of surrounding masses of nerve tissue" (Louazel).

From this time forward the relation of Morvan's disease to syringomyelia was warmly discussed. The researches of Roth (of Moscow), of Charcot, and finally of Joffroy and Achard, led ultimately to general acceptance of the view that Morvan's disease and syringomyelia were one and the same thing, and the former came to be regarded as a variety or type (Type Morvan) of the more comprehensive disorder.

On January 31st, 1890, Joffroy made a *post mortem* examination at the Salpêtrière on the body of a woman who had suffered from Morvan's whitlow, with all the characteristic symptoms belonging to it. The examination of the spinal cord was conducted under the most favourable circumstances, the absence of curvature allowing of its easy removal. There was at length an opportunity of settling the vexed question of pathology.

The existence of syringomyelia was demonstrated beyond the possibility of doubt. A cavity was found extending from the lower limit of the medulla oblongata to the mid-dorsal region, displacing a large tract of the grey matter, and the posterior columns were destroyed for a considerable distance. There was, further, a glioma of the dorsal portion of the cord, and with these peripheral neuritis of the radial and the median. "Thus was established incontrovertibly the interesting, fact till then in dispute, that syringomyelia may give rise to the clinical symptoms of Morvan's disease" (Louazel).

It may be mentioned here that Joffroy has quite recently had a case in which the *post mortem* appearances fully corroborated his earlier observation, and proved no less clearly that Morvan's disease is only a clinical manifestation of syringomyelia. In this way the pathological aspect of the matter was settled, but the original exponents of paresi-analgesia were reluctant to surrender its claims to separate recognition, and they continued to bring forward in support of their contention new arguments of a clinical nature.

Roth of Moscow had already concluded, upon purely clinical grounds, that the disease was identical with syringomyelia. It was only after the result of Joffroy and Achard's autopsy, however, that the controversy began in earnest.

It would exceed the purpose of this review to furnish a detailed account of syringomyelia. We would refer for fuller information on the subject to the very complete treatise by Bruhl, and content ourselves with the summary drawn up by Prof. Charcot (*Gaz. Hebdom.*, 1891, p. 193). It will be found to contain a notice of the principal symptoms to which we have called the reader's attention in the first part of this paper.

"1st. *Symptoms of amyotrophic paresis.*—These include muscular atrophy of the Duchenne-Aran type, gradual and symmetrical in its course, accompanied with fibrillar twitchings, and without exaggerated reflexes.

"2nd. *Disorders of sensation.*—Prominent among these is the characteristic sign for which I have proposed the name of syringomyelitic dissociation. It is not pathognomonic since it is manifested also in leprosy and hysteria, but taken in conjunction with general and progressive muscular wasting, especially where trophic derangements are also present, it imparts a highly distinctive character to the train of symptoms. The sensory affection is distributed in segments, and the muscular and special senses are not involved. Syringomyelia, like disseminated sclerosis and other diseases of the central nervous system, may be complicated by hysteria. The symptoms of the latter will in that case co-exist, and to this association is doubtless due the concentric diminution of the field of vision which has so often been met with in syringomyelia.

"*Trophic disturbances.*—Under this heading are included lesions of the skin and subcutaneous structures, ligaments and bones, depending upon dynamic or organic changes in the central or peripheral nervous system. Among such lesions, involving the skin and subcutaneous cellular tissue, are bullous eruptions, white or blue œdematous swelling of the extremities (Roth and Remak), pseudo-erysipelas, the condition known as *glossy skin*, gangrene, and analgesic whitlow, resembling that described by Morvan. Spontaneous fractures, joint affections like those which occur in tabes, spinal curvature and, finally, a special deformity of the hands, in some respects similar to Marie's acromegaly, and to which the name of chiromegaly might be applied—these are the more common disorders of the ligaments and bones met with in syringomyelia."

Morvan was naturally the least disposed to suffer the disease to which he had given his name to be absorbed in the identity of syringomyelia, and taking up the three main symptoms which belonged to it he endeavoured to shew that there were distinctions in the two cases. Whitlows, which he regarded as the essential feature of Morvan's disease, do not occur, as a rule, in syringomyelia. This is admitted by his opponents who, however, traverse the inference, urging that there is nothing more characteristic of syringomyelia than the irregularity of its symptoms; that it does not follow from the occasional absence of the whitlow that its presence is not due to that disease, and that if one symptom of a group is sometimes wanting, it is not on that account expedient to create an entirely new nomenclature for every possible combination. Moreover, though the occurrence of the whitlow is not invariable, neither is it exceptional. It has been observed in a great many cases (in four quoted by Bruhl, in one out of two by Roth, and in others by Schultze, Mader, Czerny, Joffroy, Achard and Charcot), and it has been observed as the earliest symptom of the disease. The unsymmetrical distribution of Morvan's disease, again, cannot be made to serve as a point of distinction, since syringomyelia is sometimes limited to one side or to a limb. Setting aside the question of whitlows, it needs but a glance at the general character of the trophic lesions common to both affections—lesions of the skin and of the bones, of tendons and joints—to emphasise their similarity, if not to proclaim their identity from this point of view.

There has been even more controversy on the subject of the sensory derangements, and it is upon these that certain authorities have taken their stand as "unconvinced, recalcitrant, and sceptic," to use the words of Charcot. In Morvan's disease neuralgic pains usually precede analgesia; then follows paresis and muscular atrophy; analgesia comes last. It fixes in most cases upon the hand, the forearm, and part of the arm. Sensibility is abolished not only for pain, but for touch and for temperature also. Morvan in his fifth memoir, which was written with the express purpose of refuting the supposed identity with syringomyelia, insists particularly upon this. According to him the dissociation of the modes of sensibility, so characteristic of syringomyelia, is never met with in Morvan's whitlow. The dissociation in question has been well defined by M. Charcot as a condition in which the perception of pain and temperature is lost, while the sense of touch is retained. "Some writers have gone so far as to consider this dissociated anæsthesia as the invariable and exclusive attribute of syringomyelia."

"M. Morvan has been at pains to show that his patients always exhibit some impairment of the sense of touch, and that therefore they are not instances of the dissociated sensibility which is taken to be a mark of syringomyelia."

This argument would serve if the dissociation were in fact pathagnomonic, but Charcot has expressly stated that it occurs also in hysteria and leprosy ; and Minor (of Moscow) has observed it repeatedly in traumatic hæmatomyelia. On the other hand, it has been absent in some cases of undoubted syringomyelia. Instances are on record in which sensibility to heat alone was affected, the perception of pain and touch remaining normal (Déjerine and Quilant). "Theoretically, it must be admitted that tactile sensibility may be completely destroyed as by implication of the sensory path in the spinal cord within the lesion, or by sufficiently extensive damage to the peripheral nerves." The autonomist theory is therefore met by the twofold objection that there is no symptom absolutely specific, and that the dissociation of the nerves of sensibility is not an invariable phenomenon in syringomyelia. But if this were not sufficient their contention is destroyed by the fact that the sensory disturbances, which are claimed as characteristic of syringomyelia, are also to be met with in connection with Morvan's whitlow. Some cases of the former disease exhibit only a disproportion between tactile anæsthesia and the impairment of sensibility to pain and temperature. This same condition has been recorded in many instances of paresi-analgesia (Obs. 1, 10, 19 of Morvan, Broca, Dayot, Charcot). Muscular wasting, paresis, spinal curvature, and the absence of sphincter trouble, the slow and insidious advance, are characters common to both affections. "In short, the further the analysis is carried the more completely is the artificial distinction between the two disposed of, and they are seen to pass into each other by imperceptible transitions. Each of the symptoms assumed to be specific has only a relative value. The difference resolves itself into a question of comparative frequency. On this ground it will serve a useful purpose to recognise Morvan's disease as a clinical type, but there is no warrant for rejecting syringomyelia as the underlying condition" (Achard). Bernhardt goes farther, and from a summary of the observations arrives at the following conclusions :—

(1) Clinically there is no material distinction between Morvan's disease and the affection variously described as syringomyelia, medullary glioma, and partial paralysis of sensation. (2) Whenever an autopsy has been possible spinal lesions were observed in Morvan's disease, as well as in syringomyelia. (3) From a pathological point of view the lesions are not constant and invariable, but derive their significance from the seat of the disease and from its progress, which is often extremely gradual. (4) The peripheral nerve lesions observed in both diseases are entirely comparable to those which occur in tabes dorsalis.

To conclude the account of this second period of the history of paresi-analgesia we may quote M. Charcot as reflecting contemporary opinion on the subject. "It has been abundantly proved that Morvan's disease and syringomyelia are the same clinically and pathologically. At the same time there are certain forms of syringomyelia which need to be distinguished on account of their individual characters, and of these there is one which especially deserves to be studied apart. It is but just that it should bear the name of the skilful and sagacious observer who brought it to light and gave it a clinical existence. I propose to you, therefore, to designate this form as syringomyelia, type Morvan." Thus the matter had been decided by general assent, when quite recently Morvan again came forward to claim a separate recognition for his disease. Having taken account of the work by Déjerine and Quilant he investigated the field of vision in such of his patients as were within his reach, and whom he considered to afford typical illustrations of the malady of which he makes himself the champion. He believes that he has established a notable difference as to the frequency of a constricted field of vision between these and the cases of syringomyelia reported by Déjerine and Quilant. Of eight patients three exhibited no diminution, and in three others the symptom was unilateral, while anæsthesia extended to the head. In addition to this he dwells upon the invariable occurrence of whitlow and the fissured skin, which is absent in many cases of syringomyelia. As to pathological evidence, undeterred by the autopsies of Joffroy and Achard and of Prouff, he relies upon that of Gombault, which in his view confers upon paresi-analgesia the stamp of a specific peripheral neuritis, having characters which were not found in the other autopsies, where the subjects showed the lesions of syringomyelia. In the latter syringomyelia was a secondary affection, and the cases were not examples of uncomplicated paresi-analgesia.

This, the latest, and a very recent (June 27th, 1891) protest makes it evident that the matter is not finally settled to M. Morvan's satisfaction. The only concession he is prepared to make is that "Morvan's disease may have its origin in more lesions than one. The future will show to what extent the symptoms correspond with the various lesions and whether, as I still firmly believe, there is such a thing as a Morvan's disease."

Third Period.—Dermatological. Anæsthetic Leprosy and Syringomyelia, 1891.

The third interpetation of Morvan's disease is the development of the present time. The authorities by whom it was studied, however, were struck from the first by obvious analogies, and by common symptoms which it was difficult to account for, and, as a rule, the best treatises have handled the subject of the differential diagnosis from leprosy in a manner at once so prolix and so feeble as to suggest a good deal of doubt on the subject. Thus, for instance, in Louazel's paper it is laid down that the only distinction may be the presence of leprous patches and maculæ, and that the patient's statement will disclose the exotic origin of the disease. Anæsthetic leprosy, the writer remarks, when it attacks the upper extremity gives rise to muscular wasting, paralysis and anæsthesia, the very symptoms which are essentially characteristic both of syringomyelia and Morvan's disease. The trophic derangements take the form of indolent and slowly progressive ulcers, which may be situated at the ends of the fingers—again the counterpart of the specific lesions of Morvan's disease.

Dealing with the same question in a lecture delivered on the 15th March, 1890, and published in the *Progrès Médical*, M. Charcot attempted to define the distinctions between Morvan's disease and leprosy. It is the anæsthetic form alone which presents any ambiguity. It attacks the upper extremities, causing paralysis, wasting, anæsthesia and mutilation. "It is at present," Charcot says, "a foreign disease, almost unknown amongst us. The fingers become deformed; the skin breaks and an ulcer grows in depth and extent until finally it may occupy the entire circumference of one finger, or may stop short of this. Then follows, without pain or reaction of any sort, the removal of the finger either spontaneously or by surgical interference. These events have their cause in a symmetrical neuritis described by Virchow. There is another mark peculiar to leprosy—the leprous patches or maculæ of large size scattered about different parts of the body (macular leprosy), which have acquired the name of morphœa alba or rubra, according to their appearance." In short, a diagnosis so carefully considered, and by a dualist of uncompromising convictions, relies entirely upon two points which appear to be the only ones not common to anæsthetic leprosy and syringomyelia, *viz.*, the exotic origin of the former, and the presence in it of leprous patches or spots. All the other symptoms belong indifferently to either, and a difference in their degree, if it be said to exist, is not to be depended upon for diagnosis in view of the diversity and variety so commonly affected by the peripheral symptoms in disease of the central nervous organs.

A remarkable communication made by Dr. Thibierge to the Société Médicale des Hôpitaux was the signal for a lively controversy. Dr. Thibierge exhibited a patient suffering from leprosy, with sensory disturbances resembling those of syringomyelia. He had been seen by several authorities, and the diagnosis had been much disputed. Many of the symptoms, and especially those having reference to sensation, pointed clearly to syringomyelia. In short, the case was one in which the balance of opinion wavered between the latter disease and leprosy, but M. Thibierge had the skill to direct the conclusion and the good fortune to secure Charcot's adhesion. From beginning to end of the discussion Thibierge made no allusion to the possible identity of the two diseases, between which the actual decision was so embarrassing. He contented himself with attacking one of Charcot's positions—that founded on the exotic character of leprosy —for he said, " Leprosy is to be met with in France and in Paris often enough. At present there are six lepers at the Hôpital St. Louis. In 1886 M. Leloir, in conjunction with MM. Hardy and Bernier, estimated that there were in Paris from sixty to one hundred lepers, imported from abroad. These figures do not exceed but rather fall short of the truth, and they are hardly sufficient to give a just idea of the number of patients under treatment for leprosy in the hospitals of Paris."

This statement was an unconscious step towards the unification of leprosy and syringomyelia. There remained but one of Charcot's arguments to be overthrown—that founded on the presence of macular eruptions. It was reserved for Dr. Zambaco Pacha to perform the feat. On the 25th April, 1891, he published in the *Gazette Hebdomadaire*, a statement in which he contended with evident conviction and with telling arguments for the identity of syringomyelia with anæsthetic leprosy. Morvan's analgesic whitlow is, he says, clinically indistinguishable from some cases of anæsthetic leprosy. "In both alike are seen vasomotor disturbances, multiple whitlows leading to progressive mutilation of the fingers, anæsthesia, and various trophic changes. The correspondence is absolute in all particulars, even to their experimental reproduction, between the newly discovered malady and the prehistoric disease which is wrongly believed to have disappeared from central Europe. I submit that had M. Morvan's cases been observed in a leprous country it would not have occurred to any one to make a new disease for their reception. According to the distinguished professor of the Salpêtrière, the presence of spots of pigmentation and the birth, or, at least, the prolonged residence of the patient in a leprous country, may be made the means of distinguishing leprosy from syringomyelia. "But this pigmentation is far from being a constant appearance, and the majority of the subjects of anæsthetic leprosy or Danielssen's disease, whom M. Zambaco has seen in the East, failed to present it."

The weak point in Zambaco's case is obviously the want of pathological evidence. Should it appear from the autopsy in the case of a leper affected with Danielssen's disease, that the spinal cord exhibited the lesions discovered by Joffroy and Achard in Morvan's disease or syringomyelia, there would then be no course open but to submit and once more surrender Morvan's disease as an appanage of leprosy. Zambaco holds out the prospect of an examination—pathological and bacteriological—of a cord, now in process of preparation. That should decide the matter. Whatever the truth may be M. Zambaco's conviction is on record. "To my mind the most natural inference from a purely clinical point of view is that all three (leprosy, Morvan's disease, and syringomyelia) belong to a single condition which is subject only to slight modifications, according to climate and to hygienic surroundings." It must be observed here that M. Zambaco's opinion is not adopted by Thibierge, to whom his paper was inscribed. While admitting the very close analogy from a clinical aspect between the two other morbid states and certain forms of leprosy or Danielssen's disease, he thinks that it would be premature at present to insist upon their absolute identity.

The absence of leprous tubercles in cases of syringomyelia is much against Zambaco's contention. For those who advocate the contagiousness of leprosy—but M. Zambaco is not one of them—it will be an objection that Morvan's disease has never been thought to be contagious. Leprosy induces a cachexia and stamps the features with an unmistakable character, which is never seen in syringomyelia. Finally, anatomical investigation, which has exposed the presence of peripheral neuritis in syringomyelia, has never detected in it the modes which are so prominent a formation in leprous neuritis. In this position the matter rests at present. We refrain from expressing an opinion of our own, but like Dr. Tumon, who lately published in the *France Médicale* an excellent critique on the subject, we would say, "Pathological anatomy and bacteriology have not yet spoken." We await their decision, and till it is given it is useless to speculate about the future destiny of Morvan's disease.

II.—*Syringomyelia, type Morvan*, by Ch. Achard (*Gaz. des hôp.*, July 16th, 1891).

The excavating lesion of the spinal cord, to which M. Ollivier, of Angers, has given the name of syringomyelia, has been recognised only within the last few years. The place which it occupies in the category of nervous diseases seems to grow daily with the progress of our knowledge concerning its various manifestations. It has already absorbed a great number of the progressive muscular atrophies of the Duchenne-Aran type, and without doubt it covers also many cases of cervical pachymeningitis. At the present time there is a disposition to include within its range most of the symptoms hitherto grouped as a separate disease—a disease which was first investigated by a most distinguished observer, M. Morvan, of Lannilis, and which was described as paresi-analgesia, associated with whitlows. The intimate relations between Morvan's disease and syringomyelia have attracted universal notice. The latter is extremely variable in its symptoms. It is generally characterised by a muscular wasting, most marked in the hands, but it is chiefly distinguished by a remarkable perversion of sensation, such that the perception of pain and temperature are lost, while the sense of touch remains unimpaired. Morvan's disease would appear at first sight sufficiently distinctive. Its special feature is the formation of multiple and recurring whitlows, associated with paresis, muscular wasting, and, above all, analgesia. Moreover, according to M. Morvan, tactile sensibility is always impaired.

When the matter is inquired into more closely, however, the analogy between the two affections is very striking. Thus the symptoms in both have the same localisation, namely, the upper limbs; spinal curvature is equally common in the two, and the lesions in either have the same gradual evolution. A critical examination of the supposed distinctive features will show that the trophic disturbances, and notably the whitlows, are not peculiar to the variety described by M. Morvan, but that they take place also in cases of undoubted syringomyelia. Turning to the dissociated anæsthesia again, the condition is not restricted to the latter; not only is it found in diseases of a totally different nature, as in hysteria (Charcot, Raymond), traumatic hæmatomyelia (Minor), compression of the spinal cord and nerve roots as a result of injury (Charcot), locomotor ataxy and leprosy; but it occurs also in Morvan's disease, and its existence was determined by Morvan himself in two of his earliest patients. Moreover, in the latter affection tactile anæsthesia is often very slight and limited, whilst analgesia and insensibility to heat and cold are both more extensive and more pronounced.

These considerations are undoubtedly very damaging to the position that the symptoms alluded to are of a pathognomonic character. A differential diagnosis must be based upon the most unsubstantial grounds, and the method of controversy alone tends to raise doubts against the dualist hypothesis. The opposite view, the hypothesis of identity, advocated by Roth, is sustained by very much stronger arguments. M. Joffroy and I were the first to bring forward pathological evidence in its support. In a case which presented the typical symptoms of Morvan's disease, we discovered the lesions of syringomyelia. Lately a second autopsy has corroborated the first.[1]

This is a conclusive argument in favour of the unitarian theory. Nevertheless, the anatomical observations upon which it is founded are as yet too few to warrant the neglect of purely clinical deductions. A very great interest of this kind attaches to cases in which the classical traits of syringomyelia, and especially dissociated anæsthesia, are found side by side with the special feature of Morvan's disease, namely, analgesic whitlow. Marwedel has published a case of this kind, where the onset of the disease was marked by the formation of whitlows, and where dissociated anæsthesia was present, though not complete.[2] One of Bernhardt's exhibited dissociation of a typical kind,[3] and Hoffman has described and figured a similar instance.[4]

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[1] A. Joffroy et Ch. Achard. Un cas de Maladie de Morvan avec autopsie, *Archives de médecine expérimentale*, 1er juillet, 1890, p. 540;—Syringomyélie et maladie de Morvan, *Bulletin de la Société médicale des hôpitaux*, 11 juillet, 1890, p. 640.—Ch. Achard. Syringomyélie et maladie de Morvan, *Gazette hebdomadaire*, 25 Octobre, 1890, p. 504.—A. Joffroy. Nouvelle autopsie de maladie de Morvan; syringomyélie, *Bulletin de la Société médicale des hôpitaux*, 27 février, 1891, p. 92.

[2] G. Marwedel. Beitrag zur Casuistik der Syringomyélie, *Munch. Med. Wochens.*, 18 Novembre, 1890, p. 810.

[3] A. Bernhardt. Ueber die sogenannte "Morvan's che Krankheit," *Deuts. Med. Wochens.*, 19 Février, 1891, p. 285.

[4] J. Hoffman. *Samml. Klin. Vortr.*, von Volkmann, 1891, 20.

Both these writers determine in favour of the identity of Morvan's disease and syringomyelia. Quite recently M. Charcot exhibited in his clinique a patient in whom the two symptoms, dissociated anæsthesia and analgesic whitlow, were combined,[1] and there is another such at present under the care of M. Debore. The patient in this case is a man, forty-nine years of age and a native of Paris. His personal history is very full of incident. When a soldier in Algiers he had typhus, then ague, and afterwards contracted scurvy in Mexico. He came into hospital suffering from pulmonary congestion of a tubercular character. His family history furnishes a statement of locomotor ataxy in one brother and of chorea in a younger sister. Twelve years ago he had a whitlow on his right thumb. Since that time a succession of others to the number of nine have formed on different fingers of either hand. These whitlows, or, at any rate, the earlier ones, were accompanied with intense pain. Though this is not the rule, it is recognised as occurring sometimes in Morvan's disease. The patient gives a very trustworthy account of himself, and we have his authority for a very remarkable particular. The formation of whitlow was attended with inflammation, and fever as well as pain, and the finger was much swelled up to the first joint. After the lapse of about twelve days, when he judged that the abscess was mature, the patient opened it himself with a pen-knife, and the operation was free from pain. In this may be seen one of the most characteristic features of analgesic whitlow, and there can be no doubt but that the pain at the outset was an incident of the inflammation which extended beyond the analgesic area.

The whitlows were severe, and one of them led to the destruction of the second phalanx of the thumb. Both hands are deformed. On the right side in particular the muscles are wasted, and the hand has an ape-like appearance. The nails are unequal, curved, broken and split, the skin dry and scaly. On the palmar surface the cuticle is thickened, and bears regular corns opposite the heads of the metacarpal bones. The scars resulting from the incisions made into the whitlows are also the seat of a horny thickening of the epidermis. To conclude, the hands bear the marks of injury, which, with every precaution, the patient has inadvertently sustained owing to his analgesia. Here we have a true picture of the mutilation which the hands undergo in Morvan's disease.

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[1] J. M. Charcot. Sur un cas de syringomyélie avec panaris analgésiques (type Morvan), *Gazette hebdomadaire*, 11 Avril, 1891, p. 285.

A careful investigation of the feeling power shows that the dissociation of syringomyelia is clearly marked. The sense of touch is everywhere retained, while analgesia and thermo-anæsthesia are present. In the right hand, and especially in the forearm, as high as the elbow the prick of a pin is felt only as contact. The patient is insensible of the cold of ice and of the heat of water at 70°. The same phenomena are observed on the left side, but they are limited to the palm and to the ring and little fingers. All the characters of syringomyelitic anæsthesia are present, including its segmentary localisation, without reference to nerve distribution.

It follows that the diagnosis should be at once syringomyelia and Morvan's disease. The disease is a combination of the two clinical types, or, to be more accurate, it is one of the varied forms of syringomyelia, that which in a recent paper we proposed to call the type associated with Morvan's whitlow.

In conclusion we would refer to one point worth mentioning in the case which has just been described. It is the absolute integrity of the field of vision. Now MM. Déjerine and Tuilant have regarded diminution of the field of vision as an important mark of syringomyelia, and they have produced seven cases in point. In Bernhardt's case there appeared to be some limitation of the field, at least for colours. On the other hand, M. Charcot has established the absence of this incident in fifteen cases, namely, in six of his own patients, in one whose case was published by MM. Charcot and Brissaud, in six of Roth's and two of Hoffman's. To these we might add not only the case which has been recounted here, but also another which we have lately recorded in conjunction with M. Joffroy,[1] including some others noticed by M. Brianceau.[2] The negative instances recorded amount altogether to twenty-one. On the other hand, three cases (Oppenheim, Roth, Joffroy) in which restriction of the field of vision was observed, cannot be admitted for the purposes of the discussion, since the symptom might fairly be ascribed to a co-existing hysteria. It results from a survey of all the facts that the incident in question is not of frequent occurrence in syringomyelia, and that its connection with the spinal disease is by no means proved.

James Cagney, M.D.

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[1] A. Joffroy et Ch. Achard. "Syringomyélie non gliomateuse associée à la maladie de Basedow," *Archives de médicine expérimentale*, 1er janvier, 1891, p. 60.

[2] J. Briançeau. "Contribution à l'étude du champ visuel dans la syringomyélie et la maladie de Morvan," Thèse de Paris, 1891.

Recent Papers on Peripheral Neuritis.

Morbid Anatomy. I.—*Gombault* has examined the nerves of aged subjects who had died without any symptoms pointing to derangement of the nervous system. He made in all twenty-seven autopsies; and in each he excised the external division of the dorsal digital branch to the great toe, the trunk of the posterior tibial, portions of the calf muscles, &c. The appearances presented by the first named nerve, however, are alone noted. He says that in a proportion—one half or a third—of these instances the nerves will be seen to have undergone certain changes. And since the subjects to which they belonged had not manifested during life any obvious indication of nervous disorder, their sensation in particular being normal, he concludes that the condition must be one of *latent peripheral neuritis.* The changes in question consist of a breaking up and disappearance of the myelin. The subjects examined had in all cases suffered from chronic disease, involving a protracted malnutrition of the tissues; and it may therefore be that the *post mortem* appearances belonged rather to a simple nutritive disturbance of the nerves, analagous to the wasting atrophy already described by Gombault. It might be possible to account for the persistence of sensation by assuming that the nervous lesion was less complete than it appeared to be. The hypothesis that the function of diseased fibres may be supplied by others remaining healthy is hardly applicable to the case where the loss of myelin is, so to speak, absolute. " In fact, the nerve fibres are denuded of their medullary sheath at a point more distant from their termination than usual. While they retain their functional element, the axis-cylinder, unimpaired."

II.—*MM. Pitres* and *Vaillard* have procured evidence of neuritis in persons who died a violent death, and who during life had shown no symptoms of that condition. These observers have adopted the name of *latent neuritis*, already applied to such cases by Gombault. Brissaud has established the existence of a latent neuritis in persons exhibiting the tubercular or cancerous cachexia who suffered from declared neuritis, the morbid change affecting nerves other than those which afforded symptoms during life. Moreover, the appearance in the two sets of nerves—the functionally healthy and the diseased—were identical or very similar in each case. Thus, for instance, in a tubercular patient, who had for the last two months of his life suffered from sciatica, Brissaud found very pronounced changes in the peripheral branches, both muscular and cutaneous, and in the trunk itself of the sciatic nerve; but he also found the same changes even more marked in the branches of the internal saphenous—a nerve which has no connection with the sciatic, and which during life had shown no symptoms whatever. It would follow that there is no constant relation between the symptoms which are attributed to neuritis and the morbid changes usually identified with it, since the latter may exist whether the former be present or not. The lesions in Brissaud's cases were of the most obvious kind. They presented the threefold character which is ordinarily thought to distinguish neuritis — (1) the breaking up of the myelin into globules and droplets, (2) the numerical preponderance of the smaller moniliform tubules over the larger tubules, with a continuous sheath of myelin, (3) the emptying of the sheaths. The axis cylinder, however, was intact, and consequently the lesion resembled that which Gombault has described as "segmental periaxillary neuritis." According to Brissaud there is but one form of neuritis which is capable of causing lasting damage; that is the Wallerian neuritis—a neuritis involving the axis cylinder; the other varieties, including both Gombault's segmental periaxillary form and Letulles' mercurial neuritis, being a neuritis of the myelin only. On this subject M. Déjerine has observed that degenerative lesions resembling those of Wallerian degeneration are to be found only in cases of neuritis with a more or less rapid course (toxic or infectious neuritis). In other cases where the nerve trouble is of slow development there is no reason to expect that the morbid appearances will be the same as those which can be experimentally produced. Criticising Brissaud's communication, Déjerine expressed the opinion that where such lesions as he described had failed to give rise to symptoms during life it was because they were too limited in extent; that on the other hand, where they were sufficiently developed they did cause symptoms.

III.—*Prof. Déjerine* has published a case of much interest. The patient had been affected for twenty-two months with paralysis and atrophy of his right upper extremity. The paralysis came on suddenly, as he took a pen in his hand intending to write, and it continued to be complete for several months. Sensation of every kind was absolutely suspended. The muscles wasted rapidly, and the atrophy was extreme when the case came under M. Déjerine's care. At that time sensation had become normal. The patient eventually died of phthisis eight months after his admission to hospital, and the *post mortem* failed to disclose any lesion of the brain or spinal cord.

The cellular tissue in the axilla was thickened and stained a deep brown colour. Beneath it was a mass of true bone grooved for the passage of the vessels and nerves. The cellular tissue further contained a considerable quantity of hæmatoidin granules, and at one place there was a cyst of the size of a hazel nut filled with hæmatoidin. The peripheral nerves of the part exhibited certain changes, and the cutaneous branches showed fibres in process of repair.

The neuritis in this case was due to the pressure of a hæmorrhage on the brachial plexus. Dubois (of Berne) was the first to describe an apoplectic neuritis of the brachial plexus. In respect of the diagnosis, Déjerine points out that the affection might be mistaken for an acute spinal paralysis of adults. In his own case the sensory troubles had entirely disappeared after the lapse of some months. He further believes that the symptoms of acute spinal paralysis of adults, as described by Duchenne, depend upon structural changes in the peripheral nerves, and not in the spinal cord.

Causation and Symptoms. IV.—*Oppenheim* has lately published a series of observations of a very interesting kind bearing on the subject of multiple neuritis. One case was that of an alcoholic patient, who was affected within a short period with paralysis of all four extremities, pain on pressure of the nerve trunks and muscles, and considerable œdema, especially in the situation of the adductors and in the back. In the lower limbs the paralysis was most marked in the muscles supplied by the external popliteal nerve, while in the upper extremity it was at first equally distributed throughout, and later confined to the region of the musculo spiral—the supinator longus, as in cases of lead palsy, being exempt. The most remarkable feature in the case was a neuritis of both phrenic nerves. These were sensitive to pressure and did not respond to either the galvanic or the faradaic current. There was dyspnœa, and the pulse rate exceeded 120. As a result of electrical treatment these latter symptoms, dyspnœa and tachycardia, disappeared, and there remained muscular atrophy, the more pronounced from subsidence of the œdema, partial reaction of degeneration in the peronei and an inability to flex the foot and extend the toes. The knee jerks were absent and the sole of the right foot was hyperæsthetic to a degree which made walking impossible.

In another patient, who was also alcoholic, psychical symptoms and Westphal's sign seemed to point to general paralysis or tabes. It is, however, a recognised fact that toxic neuritis is apt to be accompanied by cerebral disorder. The paralysis of the lower limbs was finally recovered from.

The third case is that of a man, aged forty, who had had syphilis twenty years before. His upper limbs were completely paralysed and his lower ones nearly so. There was paræsthesia of the hands and feet, the former were atrophied and fixed *en griffe*, there was partial reaction of degeneration, incontinence of urine, &c., and the knee-jerk was exaggerated. Oppenheim held that these symptoms could not be ascribed to the former attack of syphilis, and he referred them to the mercurial poisoning as a consequence of a long course of treatment. The motor troubles began at once to improve under the influence of warm baths and massage.

A man who was tubercular and alcoholic, was suddenly paralysed in all his limbs, the onset being attended with high fever and delirium. In the upper limbs the distribution of the paralysis conformed accurately to the type of lead palsy; confined at first to the extensors of the middle and ring fingers it subsequently involved the other extensors, while the supinators and the extensor ossis metacarpi pollicis were unaffected. Optic neuritis was also present. Death was due to paralysis of the diaphragm. The *post mortem* disclosed miliary tuberculosis and extensive parenchymatous neuritis. Oppenheim offers no suggestion as to the origin of the latter.

In a woman (alcoholic), aged thirty, multiple neuritis affecting all four limbs was ushered in by impairment of memory. The autopsy showed neuritis affecting especially the motor nerves, and in addition some anterior poliomyelitis. The latter was characterised especially by the fewness and small size of the ganglionic cells. It should be observed that in the preceding case also the spinal cord was implicated. The lesion was situated in the cervical enlargement, and consisted of a small patch on the confines of the posterior and anterior horns, in which the nervous tissue was replaced by a homogeneous hyaline substance. In this connection Oppenheim observes that it does not follow from the existence of multiple neuritis that the central nervous organs are intact. Leyden, Minkowski and Thomson have shown that the toxic agent or its product formed within the system, which causes multiple neuritis, may affect at once the peripheral nerves and the cerebro-spinal axis. The psychical disturbances which so often accompany alcoholic paralysis, enforce the view that the entire nervous system is simultaneously affected. The central lesions will differ according as it is their cellular or interstitial elements which suffer.

Bernhardt shares the opinion of Oppenheim as to the participation of the nervous system generally in the effects of toxic lesions, and he believes that this manifestation will differ both with the individual case and with the nature of the toxic agent. He rejects the possibility that a poison which is distributed throughout the system will confine itself in its action to one part only.

V.—Under the name of "puerperal neuritis" *Möbius* has described an affection which involves the nerves of the upper extremity, usually the terminal branches of the median or the ulnar or of both nerves together, and which has the ordinary characters of a neuritis. After a variable period recovery takes place. Möbius has recorded seven cases of this description. In one, which he has recently had under observation, the patient was a multipara thirty years of age. Within a month after delivery—which had been entirely uneventful—she began to complain of pain in the hand, and the flexor tendons of the middle finger were stretched like a cord across the palm and were very painful to touch. Both hands became affected in this way, and the patient could no longer use them. A fortnight later she had shooting and pricking sensations in the thighs and buttocks, and began to get weak on her legs. The hands were the only part which were obviously atrophied, and this especially in the first interosseous space. There was no anæsthesia, and the tendon reactions were obtainable in the upper extremity. The patient was able to get about, but was readily fatigued. There was also paræsthesia of the hands and feet. Some months later recovery was almost perfect, but the patient still complained of lancinating pains in the arms. The atrophy of the interossei completely disappeared.

VI.—Guinon and Parmentier adduce a series of eleven observations in which a localised neuritis of the external popliteal nerve was found to occur in the course of sciatica. The affection was marked by the sensory and motor derangements which ordinarily characterise neuritis. Disease, similarly distributed, has been known to follow injury of the sciatic nerve within the pelvis, but this is the first time in which it has been described as a consequence of sciatica. The prognosis in such a case is grave in view of the consequent atrophy and degeneration of the muscles supplied by the peroneal nerve.

VII.—The researches of Oppenheim, Siemerling, Klippel, and others have shown that peripheral neuritis is sometimes associated with cancer. Following up the investigations of these observers, *M. Auché of Bordeaux* has examined the nerve fibres of ten cancerous subjects. In every instance, except one, he discovered undoubted signs of parenchymatous neuritis. The nerve-roots and the large trunks were normal in appearance, while the terminal branches exhibited definite changes, these being most marked at the distal extremities. The changes in question affected the various nerves without special localisation.

The symptoms, when such were observed during life, were subjective sensations of cold in the lower limbs, constrictive pains in the ankle and knee, and tingling and formication in the extremities; in some cases, however, there were no symptoms to be made out. A neuritis of this kind has been observed in patients of every age, and without reference to the seat of cancer or to any implication of the meninges or spinal cord. Moreover, it has not been possible to refer the condition to pressure by the tumour, since nerves of the upper limb have been found to be damaged in cases where the cancer was situated in the stomach or uterus.

As to the causation of neuritis in connection with cancer, the only tenable hypothesis would seem to be that of auto-intoxication.

VIII.—Sherwood has published the cases of two patients who were treated for multiple neuritis in Eichhorst's clinic at Zurich. Both had been attacked some years previously with the symptoms of multiple neuritis, the right arm only being involved in one case, and both legs in the other. The first attack was followed by recovery in both cases, but relapses subsequently took place—once in the first patient, twice in the other—the localisation of the lesion being the same in successive attacks. Sherwood discusses the question as to whether the lesions were acute anterior poliomyelitis or multiple neuritis, and he concludes in favour of the latter on the ground that the patients were of mature age, and that the symptoms included pain with sensory derangements, &c.

These cases cannot be placed in the same category with recurrent attacks of toxic neuritis, where the patients are subjected a second time to the toxic influence (lead, alcohol, &c.). The cause of the neuritis in Sherwood's cases is obscure. He himself takes the view that the invariable localisation of the disease might be due to the fact that the implicated nerves were, for some inherent cause, least capable of resistance to the morbid influence.

IX.—Havage reports the case of a man, aged forty, whose illness began with fever and diffused lumbago. Three days later he complained of numbness and tingling of the extremities and weakness of the legs. As the symptoms progressed the muscular weakness was almost complete ; there was anæsthesia of the feet, and especially about the great toe, and in the same part tingling and numbness were accompanied by a scalding sensation, which the patient compared to that produced by extreme cold. There was nowhere sensitiveness to pressure. The perception of touch and pain was greatly diminished in the lower extremities, while that of temperature was but little affected. The knee-jerk was defective on the left side and completely absent from the right.

In the upper limbs there was sensory derangement of the same character and distribution as in the lower, but no anæsthesia. Paralysis affected the extensors principally. The pelvic and sacro-vertebral muscles were enfeebled. From the outset there was general wasting, but no true muscular atrophy. Finally the urine held a large quantity of albumen.

The peripheral character of the affection is evident, but its cause is doubtful. Although the patient was an inn-keeper, there was no indication of alcoholic poisoning. On the other hand the fever, albuminuria and other symptoms, would seem to point to an infectious origin. Possibly it was a case of influenza, for although evidence was wanting to show that the patient had been among its victims, the epidemic was still abroad.

On the Influence of the Spinal Trophic Centres in the distribution of some forms of Toxic Neuritis.

The *Archives de Neurologie* for March contains an instructive lecture delivered by M. Brissaud in his clinic at the Hôpital Saint-Antoine. The subject was afforded by a patient who had been for a long time under M. Brissaud's care, suffering from pseudo-tabetic alcoholic paralysis. This disease has been recently investigated by Charcot, and it is mainly owing to his researches that we are able to assign to it its distinctive and characteristic features. Briefly stated the symptoms are paralysis affecting both lower limbs in almost equal degree, and accompanied or preceded by pain. The pain is apt to be of lightning-like character, and spontaneous, and it is nearly always confined to the muscular parts, particularly of the calf. Paralysis involves especially the extensors of the leg, the dorsal flexors of the foot and the extensors of the toes, and it is rarely complete. In addition there are vaso-motor changes and disorders of secretion, with various trophic disturbances of the skin, muscles, fibrous tissue, tendons, &c. The pseudo-tabetic element in these cases is furnished by a form of inco-ordination evidenced by the gait, which is usually of that kind to which Charcot has given the name of "steppage."

Thus paraplegia, pain and inco-ordination, with an alcoholic history constitute the ordinary type of pseudo-tabetic paralysis. But as might be expected from what is known of its causation cases occur which exhibit considerable divergence from the type, and these are apt to be the most instructive, both from the difficulty which attends their diagnosis and the necessity they impose of searching out and carefully weighing the import of each symptom and of each significant circumstance.

The patient in M. Brissaud's case was a clerk, aged fifty-five, of a robust appearance. He had had syphilis twenty-five years previously, and had undergone a strenuous treatment, which stopped the course of the disease. He was a confirmed drunkard. His condition dated from 1887, and its onset was very sudden. One night he was waked by an agonising pain which he referred to a definite spot on the inner side of the left foot. This pain was of a neuralgic character, it came and went in paroxysms, and seemed to have its seat both on the surface and in the deeper parts. Since that date the patient had never ceased to experience it. It was most severe at night or in the early morning. It was apparently relieved by food, and during the day caused little trouble except when the sufferer stood about or attempted to walk. A few days after this first attack, pain, not less definitely localised, was experienced in three patches of the skin, one above the other, and all situated in a line over the internal malleolus. The new pain was less severe, but more constant than the former. The position of the painful areas was marked out by three vitiliginous patches. It appears that three small blisters had once occupied these surfaces. Vesication was followed by a very slow process of healing, and the spots in question were for a long time the seat of ecchymosis. In the course of a few months the colour disappeared, and the site of the blisters was to be distinguished from the surrounding skin by the absence of pigmentation. On this point M. Brissaud observes that the event is the very opposite to that which ordinarily takes place—the effect of a blister being increased pigmentation. "Leucoderma is the consequence of depigmentation. In the great majority of cases, if not invariably, it is evidence of trophic nervous disorder. It is known as a consequence of severe neuralgia, and it then" (as in this case) "occupies the spots where antecedent pain was most intense. What it is of importance to notice, however, is that the leucoderma assumes the exact shape and dimensions of the blisters applied three years ago, and that it was represented formerly by similar patches of ecchymosis." At the time of which we write these patches were no longer the seat of pain, they were insensible, or nearly so, to touch, pain, and temperature.

Difficulty in walking began about the same time as the pains. It was gradual in its development, commencing with mere awkwardness in both legs. The condition progressed in the left, and slowly disappeared from the right. A year after the onset of the disease the patient described his mode of walking thus: "At times I would lift my left leg as if I were walking over eggs, and could not help it; at others I used to drag it after me, as if it did not belong to me." This continued to be his mode of progression and commenting on it, M. Brissaud points out that when the patient raises his leg as if "walking on eggs" he is a "stepper," when he drags the limb after him, he does so because it is paralysed. Enquiring more particularly as to the distinction between the two gaits he maintains that it depends only on the functional efficiency or exhaustion of the quadriceps extensor, by which the "stepping" movement is accomplished. It is curious to note that the patient was in this respect, as in that of pain, worst in the morning, and it was the usual accompaniment of fatigue. The fact that his ability to "step" varied also from day to day suggests the possibility of a varying influence, depending probably upon the quantity of drink taken. The symptoms as described, though subject to occasional amendment and exacerbation, progressed on the whole from bad to worse during a period of two years. At the end of that time the left leg was diminished in bulk, but some of the muscles were more atrophied than others. Those most affected were the adductors, both vasti, the rectus femoris, the extensors in the leg, and the gastrocnemius. The left knee-jerk was absent. When the patient was made to stand erect it was observed that the left patella was on a lower plane than the right, thus indicating the weakness of the quadriceps. "We may here direct our attention to a fact, which, though it has not much importance from the point of view of diagnosis is yet not without its interest. You may observe, just above the left patella, a muscular fold directed obliquely from above downwards and inwards, and causing a prominence on the inner side of the thigh. Is this a normal appearance? For it does not occur on the sound side. You have here an occasion for reference to your anatomical knowledge—not to the knowledge which you acquired as students—for the present purpose it is already obsolete—but to impressions of more recent date. We have to do here with *surface* anatomy, the science which deals with the living body, not the cadaver—with what may be seen or made out through the skin when the muscles contract, not with the relations of lifeless parts as they are taught by dissection. This department of anatomy was not created for artists; of that you have a proof before your eyes, since we are now concerned to enquire what is the meaning of the muscular prominence just above the patella. M. Richer, who has lately devoted an admirable work to the subject of surface anatomy will provide an answer to the question. The prominence alluded to is called the supra-patellar fold of Gerdy. It is the lowest fleshy portion of the vastus internus, which "forms a prominence distinct from the rest of the muscle in certain states, and in given anatomical postures." (Richer). It occurs during muscular relaxation, and it is more evident as the latter is more pronounced. When the muscle contracts the fold diminishes, and in some persons disappears altogether. The anatomical cause of the supra-patellar fold of Gerdy is to be found in a transverse band of the fascia lata which passes across the front of the thigh in its lowest part, and is connected internally with the internal intermuscular septum. Beneath this the lower part of the vastus internus protrudes in the manner of a hernia, and the situation of the band corresponds to a groove on the surface which separates the position of this part from the remainder of the muscle. "The interest attaching to this groove," says M. Richer, "is very great, because depending upon the same cause as the prominence below it, namely muscular contraction, but muscular contraction in another direction, it is evidence of the physiological condition of the vastus internus, and it is eminently a part of living anatomy." M. Brissaud points out that this sign has a pathological as well as a physiological value. It is a measure in this case of the relaxation of the vastus, the weakness and flaccidity of that muscle being insufficient to draw its lower fibres above the level of the constriction. Intimately connected with this is the dropping of the patella already noticed. In concluding a reference to the muscular symptoms it must be mentioned that the glutei alone of the posterior groups had escaped atrophy, and upon this fact M. Brissaud bases important inferences.

Turning now to the trophic lesions we are told that there was a herpetic eruption localised in two places. One of these was on the back in the line of the iliac crest, the other on the front of the abdomen below and a little to the left of the umbilicus. The eruption therefore coincided with the distribution of the ilio-hypogastric nerve, the first branch of the lumbar plexus. Its occurrence had been marked by an aggravation of the pains and paralysis, and it is important to bear in mind that it was not the only instance of trophic derangement in the course of the disease.

Reviewing the symptoms as a whole it will be seen that they all fall within the regions supplied by the branches of the lumbar plexus. The earlier pains were in the district of the internal saphenous; the paralysed and atrophied muscles were supplied by the anterior crural, the external popliteal and the obturator. The glutei, which were not implicated, are supplied by the small sciatic, a nerve derived from the third and fourth sacral roots, and its origin is therefore below the position of the presumed lesion. It is to be noted also that the severer symptoms may be referred to a still narrower compass, lying entirely below the level of the second lumbar nerve.

To sum up, M. Brissaud remarks that the symptoms pointed to a neuritis in so far that they were diffused over a wide extent of surface; but the diffusion perhaps is only apparent since all can be referred to a definite part of the lumbar plexus, and possibly to a situation still more central, in the part where the implicated nerves take their origin from the spinal cord. He proceeds to weigh the reasons for and against a possible central lesion, and as a preliminary step he considers the grounds upon which locomotor ataxy may be excluded.

With this view he discusses the character of the inco-ordination. Frankly stating that ten years ago the disease would certainly have been mistaken for one of tabes, he rejects the conclusion definitely on this ground alone: "The patient does not fling his leg forward and bring his heel to the ground. He 'steps.' His peculiar gait depends upon paralysis of the extensor of the leg, and to the same cause is due the absence of the left knee jerk. Its persistence on the right side, where, however, it is impaired, is also significant. The care with which the patient follows his movements with his eyes is not after the manner of a tabetic; it is determined merely by a sense of insecurity in walking." There are points of distinction also in connection with the pains in alcoholic pseudo-tabes. In character they hardly differ from lightning pains, but they exhibit the morning exacerbation which belongs to alcoholic neuritis.

Ataxic pains have no periodicity, and they arise at any hour of the day or night.

It is, doubtless, true that locomotor ataxy occasionally declares itself by a very few morbid signs, but among these there are always one or more which are pathagnomonic, such as the Argyll-Robertson pupil, myosis, mydriasis, or inequality of the pupil, and transient paralysis of the ocular muscles, Romberg's sign, &c. None of these had taken place here. On the other hand, it is necessary to allude to certain psychical peculiarities of a kind which, according to Charcot, are almost inseparable from alcoholic paralysis. Among these were extreme depression and a fixed belief that the case was hopeless, inducing a contemplation of suicide, and with this insomnia or restlessness in sleep.

Again, it is a point to be weighed in diagnosis that locomotor ataxy is essentially a progressive disease, while M. Brissaud's patient has himself borne witness to his "ups and downs," which were determined, no doubt, by the vicissitudes of the alcoholic habit.

M. Brissaud considers it not worth while to anticipate objections that may be based on the fact that the paralysis was unilateral, or on its limitation to a definite nerve distribution. He contends that, while paraplegia is the rule in alcoholic paralysis, the exceptions in which the disease has been more limited and even eccentric in its incidence, are sufficiently numerous to justify the expectation of extreme divergence from the type in a few instances, and that it is not matter for astonishment if a case presents itself in which the symptoms are limited to the distribution of a plexus or of part of it.

That the disease was a neuritis he thinks was sufficiently declared by the occurrence of zoster, added to the earlier manifestations. He then devotes himself to a consideration of what he professes to be at once the most interesting and the most difficult topic—the causes which determine the seat of the inflammatory lesion in the lumbar plexus. For this purpose he takes up the analogy of lead palsy. Indicating the immunity enjoyed by the supinator longus, he points out the difficulties which have to be met in attempting to explain it. The expectation that a spinal lesion would be found to fit the case has not been justified in general by events; nevertheless, such lesions have been discovered by Friedländer, Oeller, Hallopeau, Renaut, Vulpian, and Oppenheim. Moreover, negative instances from the nature of things have not the same value as positive, and Charcot has laid it down long since that even if lead paralysis does not depend upon subacute anterior poliomyelitis, as was at first too hastily assumed, it is not on that account impossible that an affection of the spinal cord has much to do with the neuritis, even though that affection may not be altogether comparable to a poliomyelitis. The same considerations may be obviously applied to other forms of toxic neuritis.

The combination of sensory, with motor disturbances in the case of alcoholic paralysis, affords additional evidence of a peripheral cause, and in those cases where the parts attacked are supplied by nerves which are in every way remote from one another, it is impossible to suppose otherwise than that the poison has fastened upon their terminal parts in a manner, as it were, at random. But where, again, the implicated nerves can be traced centrally to a point where they all converge within a limited space, it is then, M. Brissaud contends, more reasonable to suppose that the poison has taken effect at that limited part. Lendet has reported a case of alcoholic paralysis in which only the muscular branches of the ulnar were involved. Here it may well be assumed that the spinal nuclei of that nerve were the original point of attack. Again, in the ordinary paraplegic form of that disease a toxic irritation of a particular spinal segment is most adequate to account for the symptoms. Finally, in the case under discussion, the simultaneous and exclusive implication of nerves which take their origin from a definite and limited portion of the spinal cord seems best accounted for by a primary lesion situated in that part.

Such speculations are not entirely against the weight of evidence. Autopsies are rare in alcoholic paralysis, but mostly they have disclosed changes in the spiral cord. Ættinger has observed vacuolation of the cells in the anterior horns. Finlay (reported in the Royal Medical and Chirurgical Society's Transactions) and Sharkey (Pathological Society's Transactions) have described a diminution of the grey substance and changes in the cells, and their observations have been confirmed by Korsakoff.

Granting, however, that such experiences are exceptional, we are not warranted in the inference that because the more highly specialised nerve structures are not obviously damaged, we must reject the possibility that the irritating or degenerative process in toxic peripheral neuritis is the effect of a spinal cause. Witness the constant application of the word "dynamic" in reference to nervous effects. This word is used to characterise certain structural changes in the trophic centres, such as with our appliances we have not the means of recognising. Hysteria is *par excellence* the sphere of dynamic influences; but without wandering from the present subject an instance may be chosen in which this undetermined principle is commonly made to exercise control over the incidence of peripheral neuritis. "If there is a fact universally admitted it is the trophic influence exercised by the large motor cells in the anterior horns over the nerve fibres to which they give origin, and through them over the muscular fibres. The chief symptom of anterior poliomyelitis is muscular atrophy—it is, so to speak, almost indispensable. The result of that condition, whether primary or secondary, is wasting of the muscle fibres. What I have to say here concerns only the secondary forms, such as arise in the course of systematic spinal lesions, of which I will instance only locomotor ataxy and degeneration of the pyramidal tract as a consequence of hemiplegia. In ataxic and hemiplegic subjects circumscribed muscular atrophy occasionally takes place, and *post mortem* examination reveals marked changes in the cells of the anterior horns. To confine myself to the case of hemiplegia; here the muscular atrophy sets in at an uncertain stage, and always, it is to be observed, in the muscles which exhibit the highest degree of contraction. In such cases both M. Pitres and I have observed well marked degeneration in the corresponding motor cells . . . . and it would seem to result that the muscular atrophy is immediately dependent upon an anterior poliomyelitis, which in its turn is caused by the degeneration of the pyramidal fibres. This conclusion, however, is negatived by later experience. In conjunction with my colleague, M. Déjerine, I have reported cases in which hemiplegia was followed by muscular atrophy, while the autopsy showed no trace of poliomyelitis. The nerves, on the other hand, exhibited all the characters of parenchymatous inflammation. How is this fact to be explained? Obviously the atrophy was caused by the neuritis, but on what did this in its turn depend? Clearly upon the lesion which gave rise to contracture, since both atrophy and neuritis were most marked in the muscles, which were the seat of contracture. Now the lesion which caused the contracture is the degeneration in the pryramidal tract; but the pyramidal tract contains not one twentieth of the fibres represented at the periphery by the branches and muscular twigs derived from the spinal cord. To explain the influence of a descending degeneration upon the peripheral neuritis of hemiplegics it is imperative to admit the intermediation of the grey matter which connects those nerves with the pyramidal fibres."

From these considerations M. Brissaud concludes that alcoholic neuritis is not always in the strict sense peripheral, that while the peripheral nerves in some cases alone suffer from the toxic agency the spinal centres do not always escape, and that where the distribution of the symptoms is such that they can be referred to a central lesion it is more logical to seek their explanation upon the same principle as is applied in the case of other diseases of the nervous system.

JAMES CAGNEY, M.D.

Poliomyelitis and Multiple Neuritis.

The *Nouvelle Iconographie de la Salpêtrière* contains an important paper by MM. Paul Blocq and Marinesco. It is devoted to an exposition, on the one hand, of the evidence upon which rests a belief in the existence of acute and subacute anterior poliomyelitis of adults; and on the other, of the probable relations which those affections bear to multiple neuritis. Premising that the whole question is yet far from settled the writers refer to the recent works of Raymond, Strümpell, and Déjerine-Klumpke as setting forth in the fullest manner both the actual state of knowledge on the subject and the varying tendencies of opinion. That of the writers is very clearly in the direction long ago marked out by Duchenne, and their task resolves itself into an uncompromising support of his classification. To this end they detail the symptoms and the *post mortem* appearances of three cases reported respectively by Rissler, Schültze, and Oppenheim, with an account of the autopsies in each and a commentary, which adds greatly to their value. These cases appear to have been selected because, in the writers' opinion, they bear most closely upon the matter in dispute, and it is maintained that they support in a very conclusive manner the two-fold contention; first, that there are such diseases as acute and subacute, or chronic poliomyelitis of adults; and, secondly, that those diseases have the characters speculatively assigned to them by Duchenne—characters, that is, which imply an obvious relationship with acute infantile paralysis. In connection with Rissler's case, which was of the acute disorder, mention is incidentally made of the fact that it further supports the view held by Charcot, and opposed by Roger, Damaschino and Roth as to the minutiæ of the morbid anatomy in anterior poliomyelitis. Long before an opportunity had arisen of inspecting the recent lesion M. Charcot had hazarded the opinion that the essential change at the beginning of the disease would be found to be one implicating the Ganglionic cells, and not as his opponents believed the vascular and insterstitial tissue. Rissler secured a *post mortem* within seven days of the onset of symptoms, and the appearances fully justified M. Charcot's expectation. The fourth observation is published here for the first time, and it possesses a very great interest, not only as bringing into prominence the fundamental distinctions between the atrophy of neuritis and that of anterior poliomyelitis, but as a typical and well authenticated narrative of the clinical events, which belong to the former condition, illustrated and supported by an admirable autopsy. The latter was conducted by the writers themselves. The case during life was under the care first of M. Hayem and afterwards of Professor Charcot. The illness had extended over a period of twenty years, and had undergone remissions and relapses of the most acute character. At the outset, as was inevitable at that early period, it had been regarded as an acute spinal paralysis. The attack had been ushered in by fever, there was generalised atrophic paralysis, and this was succeeded by local recovery—a clinical history, which then admitted of no other explanation.

MM. Blocq and Marinesco indicate the principal points of distinction. (1) The gradual invasion—the earlier symptoms had reference to a general disturbance of nutrition. Prominent amongst them were digestive troubles, and they held the field for eight days before paralysis occurred. (2) Psychical phenomena, such as maniacal delirium, sleeplessness, nightmare. (3) The fact of relapse, in which the invasion stage was repeated with all its original severity. As favouring the presumption that the case was really one of multiple neuritis attention is directed to the general cachexia, with diarrhœa and vomiting, delirium, œdema, and vasomotor derangements.

Even more instructive are the results of the autopsy. The lesions were found to be extremely pronounced in the muscles, very limited in the nerves, and almost absent, or merely senile changes, in the spinal cord. Degeneration of muscles was most complete and decisive; the nerves affected were almost exclusively the intra-muscular branches. This then was a case of generalised muscular atrophy, depending upon a neuritis, and distinguished by clinical incidents of a very definite order. These are considered at some length in the final chapter. The whole question of psychical disturbances in connection with multiple neuritis is made the subject of a critical review, and reference is made to a recent work by Korsakoff, dealing with a class of cases to which he has given the name of "polyneuritic psychosis." The affection consists anatomically of the lesions of multiple neuritis; and clinically of symptoms referable, on the one hand, to the extremities, and on the other to the cerebral function. Korsakoff has collected sixteen cases, in most of which there were a sudden invasion with fever, abdominal pain, vomiting and prostration, then excitement and delirium accompanying amyotrophic paresis of paraplegic distribution, or extending to all four limbs. There is nothing more regular than the association between multiple neuritis of toxic origin and disturbances of cerebral function. Some further allusion to the subject will be found in the report of a clinical lecture by M. Brissaud. MM. Blocq and Marinesco remark that in their case, as in many others not obviously ascribable to a toxic agency, the cerebral symptoms seem out of proportion to such a cause as a primary peripheral neuritis. So far as the reflection is founded on the consideration of their own case it is obviously weakened by some doubt as to whether alcoholic poisoning can be excluded. But in general it has much pertinency, and the association is giving rise to a good deal of discussion. On the one hand there is a growing tendency to regard all forms of peripheral neuritis as of toxic origin. With the extension of this tendency the explanation of Strümpell and Raymond becomes at least more adequate. These observers hold that the toxic or infectious agent may fasten at once upon any or all the parts represented by the ganglion cell, the motor nerve-fibre and the muscle-fibre. For suggestive remarks upon this subject the reader is referred to the paper by M. Brissaud, already alluded to. Blocq and Marinesco, however, take occasion to observe that the neuromuscular arc has a greater complexity than the statement reckons with. To the structures enumerated must be added the corresponding part of the cerebral cortex, and they claim for it a leading *rôle* in the dispensation of trophic lesions. Babinski has reported a case of hemiplegia with muscular atrophy, in which the absence of spinal or neurotic changes has led him to infer that it was the cerebral lesion which caused the atrophy, and Richet has reported analogous appearances in dogs. An argument based upon the frequent occurrence of atrophy in the so-called psychical paralysis of hysteria is perhaps not very incisive. It is basing an inference on a very unstable foundation. More satisfactory is a reference to experiments performed by Raymond. Observing the rapid progress of degenerative changes in the muscles of newly-born animals in which the pyramidal tracts are not yet developed, Raymond performed section of the tracts in adult animals, and obtained the same result. This he attributes to withdrawal of the cerebral influence.

The authors conclude with the following summary of the inferences drawn from their researches :—

1st. There are morbid states which correspond exactly in their clinical features to the disease described by Duchenne under the name of acute and sub-acute spinal paralysis of adults. These morbid states depend, as Duchenne had foreseen, upon lesions of the anterior horns of the cord.

2nd. On the other hand there are groups of symptoms which so closely resemble these as in some cases to be confounded with them, and in which no appreciable lesions of the spinal cord are found.

3rd. It is impossible to affirm definitely that the disorder in these latter cases is dependent upon the co-existing peripheral neuritis.

4th. The clinical phenomena go to prove that the cerebral centres are implicated in the process, and in our opinion there is ground for the belief that they take a part in its production (amyotrophic polyneuritis associated with psychomotor disturbance).

5th. It does not appear to us possible, at present, to determine the relationship between the amyotrophic paralyses of poliomyelitis and those of multiple neuritis. The hypothesis which commends itself most to our judgment is that of a lesion of the neuro-muscular arc (motor cortical cells, ganglion cell of the cord, motor nerves and muscles) effected by a pathogenic agent which may act with greater or less virulence upon any part of the arc.

James Cagney, M.D.

Ballet on Hysterical Attacks Simulating Partial Epilepsy (Gaz. des Hôp., July 16th, 1891).

The writer points out that the initial phase in an attack of hysteria major bears a close resemblance to an epileptic fit, and on this account it is distinguished by Charcot as the "epileptoid" stage. He proposes to shew that hysteria may also imitate the lesser or Jacksonian epilepsy, giving rise at one time to spasms of limited extent, at others to convulsive seizures of the whole of one side of the body. He exhibited a case of the latter kind. The subject was a young girl who had hemianæsthesia of the left side, and who had several times suffered the "grand attack." She was therefore the victim of hysteria major.

To the typical features of the grand attack there had been added in the case of recent seizures certain other manifestations. To elicit these it was only necessary to stroke the patient gently over one of the two hysterogenic areas which she presented. These were respectively under the left breast and on the top of the head. The immediate effect was spasmodic contraction of the sternomastoid of the right side and of the trapezius on the left, the head inclined to the left side and the patient foamed at the mouth. At the same time the left side of the face, or that part of it supplied by the inferior division of the facial nerve was thrown into contractions. This group of symptoms resembled closely, and might be mistaken for a cerebral lesion causing irritation of the motor centre for the face. Such a lesion would be situated at the base of the right ascending frontal and parietal sonvolutions.

M. Ballet remarks that the possibility of such an attack must be recognised in order to avoid serious mistakes of diagnosis, and he relates an instance in which he himself fell into an error of the kind. The patient had been under his care for some time, suffering from hysterical disorders. On one occasion she was, in his presence, seized with epileptiform convulsions which had their starting point in the leg. For twenty-four hours he continued to believe that the case was one of tubercular meningitis—an impression which was borne out by some of the facts observed. Presently, however, he discovered his mistake, and recognised that he had to do with a hysterical phenomenon. He points out further, to emphasise the readiness with which a false conclusion may be formed, that, at the time of this occurrence, he was already familiar with the hysterical form of partial epilepsy, and had written a monograph on the subject.

The circumstance of all others most misleading in these cases is found in the fact that the hysterical attack may stop short with the epileptiform stage. It may happen, for a time at least, that other phenomena (*grands mouvements*, emotional attitudes, hallucinations) are wanting to assign to the attack its true hysterical character. This is generally due to the fact that the seizures follow one upon another without intermission, and with such rapidity that these constituent phases have not time to develop. The result, to all appearance, is a condition, not indeed of epilepsy, but of epileptiform convulsions.

In the case under discussion the patient had had sixty-two attacks in the space of an hour and a-half. Ordinarily they were separated by a variable interval, but they could readily be brought on at any time. When one was about to take place, and before the facial spasm set in the pupils were depressed, the eyes converged towards the root of the nose, and the limbs assumed a cataleptic state, retaining whatever posture they were put in. The cataleptic condition persisted throughout the epileptiform stage, and for some time afterwards. Frequently, as might have been expected, facial convulsion was attended with hallucinations. When this order is observed diagnosis becomes comparatively easy. When, on the other hand the seizures are repeated in series, mistakes are readily made. Even under these circumstances, however, there are marks by which the hysterical may be distinguished from the true epileptiform attack. The chief is the absence of any rise of temperature, which (Charcot, Bouchard, Bonneville) invariably accompanies a succession of epileptiform convulsions.

Another point is the immunity of the implicated limbs from subsequent paralysis. Temporary paralysis is often observed as a consequence of Jacksonian epilepsy; it is very rare in connection with the partial epilepsy of hysteria. It would be a mistake, however, to suppose that it never occurred. Again an important inference may be based on an analysis of the urine voided after a fit, since it has been shown by Gilles de la Tourette and Cathelineau that urea is increased after an epileptic, diminished after a hysterical attack. In the latter case also it has been stated that the amount of earthy and of alkaline phosphates respectively tends to become equal. This, however, is not always so.

M. Ballet further remarks that, whereas in the epileptic stage of hysteria major, as in an epileptic fit consciousness is lost, it may be retained in hysterical attacks simulating partial epilepsy, just as it is retained in a fit of Jacksonian epilepsy. In illustration he pointed out that the patient then under examination was apparently alive to all that took place. She habitually asserted that she could follow the conversation around her; while her face underwent contortions she would obey the instructions given to her, such as to bend her arm, or to kneel down. In fact she was in some respects more docile than when in her normal state of health.

JAMES CAGNEY, M.D.

On the Etiology of Tabes. By Prof. W. ERB, Berlin, Klin. Woch., July 20th and 27th, 1891.

Professor Erb states that since his last publication on the subject, in 1883, he has been constantly engaged in collecting a larger number of exact observations for the study of the etiology of tabes and its connection with syphilis. Since he published his first papers on the question, in 1879 and 1881, he has had the satisfaction of seeing the accuracy of his statistics confirmed from nearly every side, and the conclusions on the relation between syphilis and tabes, which he then drew, almost unanimously accepted even by his former opponents.

After briefly dismissing some objections which have been made against his statistical results, he proceeds to criticise a report of the German Army Medical Department, in which it is stated that, in cases of tabes dorsalis occurring in soldiers, only 7.4 per cent. had previously been infected with syphilis (*Sanitatsbericht über die Deutschen Heere im Kriege gegen Frankreich*, 1870-71, Bd. vii. B. S. 312-391). From a consideration of the data upon which this report is based and of the circumstances under which it was drawn up, he is of opinion that the conclusions arrived at in it cannot be received as correct, and he compares them with the results of the examination of a series of fifty-one cases of tabes in military officers occurring in his own practice, selecting officers because they are more likely to give reliable information on the points in question than private soldiers. Forty-nine of these patients had previously acquired syphilis and one of the two remaining had had gonorrhœa; to say the least there is a striking contrast between the two results.

Passing on to the analysis of his own cases, Professor Erb has since 1883 collected 370 new observations. Of these 370 patients, 300 belong to the upper classes and come from his private practice, whilst 50 were seen in hospital practice and are drawn from the lower orders. There were in addition 19 women. For the purpose of his statistics he selects the 300 patients belonging to the better classes, for the reason that they are much more likely to afford accurate information on the points in question. Of these patients, who were thoroughly and carefully examined with especial regard to the connection between syphilis and tabes, in 33 or 11 per cent. there was no apparent syphilitic infection, in 267 or 89 per cent. there had been previous infection, and of the latter, 190 or 63.3 per cent. had well marked secondary symptoms, 77 or 25.7 per cent. a chancre only. Of the 33 non-syphilitic cases, a large number must be regarded as doubtful rather than certain; not fewer than 19 showed suspicious symptoms, rashes, scars at the angles of the mouth, on the tongue and on the penis, or had been treated by their medical men for sore throat, or had wives who had given birth to still-born children or had aborted; 24 of these patients had had gonorrhœa, some many times, and some an obstinate gonorrhœa or a stricture. These 24 patients must have run the risk of exposure to the syphilitic poison, and can only be considered as probably free from infection. In the remaining nine cases, three per cent., no evidence of syphilis could be obtained, but it should be said of them, not that they were certainly not syphilitic but that there was no clear trace of syphilis. Of the 77 cases with a chancre only, in 15 it was described as hard, and 31 were treated with anti-syphiltic remedies, so that the majority of these patients are to be counted as syphilitic. These statistics are in complete accord with Erb's earlier ones, and adding them all together there are 500 cases of which 89.2 per cent. were syphilitic, and 10.8 per cent. non-syphilitic. The 50 cases from the lower classes gave 12 or 24 per cent. as not infected, and 38 or 76 per cent. as previously infected with syphilis; in 26 of the latter syphilis was certain, while 12 had a chancre merely; the number of the non-infected is here probably stated higher than is actually the case.

As to the time of onset of tabes after syphilis had been incurred; in the above 300 cases, this took place at from 1.5 years in 12.3 per cent.; 6-10 years in 37 per cent.; 11-15 years in 24.7 per cent.; 16-20 years in 14.2 per cent.; 21-25 years in 4.8 per cent.; 26-30 years in 1.9 per cent.; 30-35 years in 0.7 per cent.; not ascertained in 4 per cent. This agrees with former statistics, the onset occurring in the majority of cases from 6-15 years after syphilitic infection, and within 20 years in no less than 8.8 per cent. Erb has also endeavoured to discover in what proportion patients who came to him for all kinds of complaints had previously had syphilis. He has investigated this question on 5,500 men, mostly drawn from the upper classes, and he finds that 77.5 per cent, had not had syphilis, while 22.5 per cent. had had it at some former time: of the latter 12.1 per cent. had suffered from secondary symptoms, 10.4 per cent. from a chancre only. It must be remembered that these figures do not apply to the general healthy community, but only to men who came to him as patients suffering from various complaints. It follows, therefore, that in tabes 89.2 per cent. of the patients have formerly had syphilis, while in other diseases only 22.5 per cent. have suffered from it. Erb concludes that syphilis plays a very prominent part in the causation of tabes, and that almost only those persons run a risk of becoming tabetic, who have formerly incurred syphilis.

Of the 19 women 9 were drawn from the upper, 10 from the lower classes; 9 or 47.4 per cent. of these were certainly syphilitic; 8 or 42.1 per cent. almost certainly so, and 2 or 10.5 per cent. were not syphilitic. The proportion for women, therefore, closely corresponds to that for men.

For the determination of the other ætiological factors of tabes, Erb takes 281 (of the 300) cases in which special effort had been made to discover them. The following result appears. In 27 per cent. syphilis was the only morbid influence at work, in 11 per cent. it was associated with exposure to cold, in 6 per cent. with overwork, in 9.6 per cent. with sexual excess, in 5 per cent. with injuries, in 12 per cent. with a neuropathic heredity, in 13.5 per cent. with both exposure to cold and overwork, in 1.7 per cent. with exposure to cold and sexual excess, in 7 per cent. with overwork and sexual excesses, in 4 per cent. with more than 3 of these injurious influences.

Of the 281, in 32 cases in which syphilis was absent exposure to cold and sexual excess occurred in a slightly greater percentage than the other causes. Syphilis, therefore, appears to be undoubtedly the most frequent and potent morbid process that precedes tabes, while the other factors only very seldom cause the disease when acting alone, but in the large majority of causes act in conjunction with syphilis.

As to the question of direct heredity, in 2 out of 275 cases the fathers of the patients had tabes; in the first instance the son had not had syphilis, and its occurrence in the father could not be ascertained; in the second both father and son had been infected with it.

In two families two brothers were affected in each, the tabes was preceded by syphilis in all the patients. Direct inheritance or the transference of the disease from father to son plays no part therefore in the etiology of tabes. As to the presence of a neuropathic heredity this was present in 77 out of 271 cases or only 28 per cent., and taking into consideration the great frequency of a neuropathic taint in our days in the upper classes, it would, therefore, seem to have no important influence on the occurrence of tabes: on the other hand the proportion is high enough to make it likely that it acts in co-operation with other causes. Similarly neurasthenia is only of importance as an ætiological factor when it occurs in combination with the syphilitic infection, and not when present alone. Exposure to cold and overwork also seem to have by themselves little importance, but again in conjunction with syphilis account for 31 per cent. of cases.

In 43 out of 271 cases or 15.8 per cent. sexual excess was noted. Erb thinks that this indicates that sexual excess plays a more important part than current opinion attributes to it.

In 27 cases sexual excess occurred together with syphilis, in 3 alone, and in 7 with exposure to cold and overwork as the determining causes of the disease. His researches have gradually brought him to consider that in not a few cases sexual excess is a decidedly active cause, but almost exclusively in those, who have at an earlier date been infected with syphilis; in 8 men the first symptoms of tabes came on soon after marriage, and this is especially apt to occur where elderly men of about 50 years of age have married again, and exerted themselves beyond their strength.

With regard to the influence of occupation upon the incidence of tabes; of 550 cases, 237 were merchants, 27 manufacturers, 50 officers in the army, 39 railway officials, architects, engineers, &c.; 34 judges, attorneys, &c.; 26 medical men, 24 professors, teachers, chemists, apothecaries, &c.; 20 landlords, 19 innkeepers, brewers, &c.; 13 fundholders, 1 a clergyman, 42 mechanics, 30 labourers, &c.; 18 gensdarmes, soldiers, fishermen, engine-drivers, &c. The large proportion of merchants and officers affected by the disease is very striking. Whether this stands in relation to the percentage of these classes in the population, or whether they are affected with syphilis in greater relative proportion Erb does not determine, but thinks that the latter is most probable, and in connection therewith points out that only one patient was a member of the clergy, and he had had syphilis.

In three cases the onset of tabes occurred at an unusual age, and in all three syphilis was also acquired unusually early or late; one man acquired syphilis at the age of 57, and developed tabes at 66; another had syphilis at 54 and tabes at 59; and a third acquired syphilis at 19½, and tabes at 22 years of age.

In four cases tabes developed whilst unmistakable syphilitic lesions were still present.

In three instances husband and wife were first both infected with syphilis, and later both became tabetic.

Professer Erb has found that one single severe exposure to cold may precede the onset of tabes, and thinks that prolonged residence in damp places, working in offices with cold floors, sleeping in damp and cold dwellings are efficacious as causes, but almost without exception only in those people who have previously had syphilis.

To conclude, syphilis is by far the most frequent and potent cause of tabes dorsalis, and by the side of it other morbid influences play a very subordinate rôle. The further supposition which has been maintained by Moebius and Hirt that tabes must be referred to antecedent syphilis in every case, even when there is no proof at all of past syphilis in a portion of the patients, is not supported by Erb's investigations, for cases are much too numerous in which the most searching investigation fails to show a trace of syphilis, either hereditary or acquired.

On Atrophy of the Optic Nerve and its value in Prognosis in Sclerosis of the Posterior Columns of the Spinal Cord. By Dr. JOHANNES MARTIN.

The fact that where there is atrophy of the optic nerve in Tabes Dorsalis the other symptoms of the disease remain stationary at an early stage, or attain only a slight degree of intensity has been remarked by many observers, since it was first pointed out by Benedikt of Vienna, and the object of this memoir is to give more exact information on the antagonism which exists between atrophy of the optic disc and disorder of co-ordinated movement. Dr. Martin gives a summary of the writings of various authors on this subject, and states that his own observations confirm the statements of Benedikt, as to the influence of atrophy of the optic nerve on the development of tabes, in the pre-ataxic period. It is very rare for a tabetic patient, the subject of amaurosis at an early stage of the disease to become ataxic, while in many cases, lightning and other pains become less severe after amaurosis is established. On the other hand he has not found that disorders of movement, if once well marked, improve with the subsequent development of optic atrophy. As a matter of fact amaurosis is rare in patients who have reached the ataxic stage, but in cases where it has occurred, no improvement in the disorders of locomotion has been noticed.

The patients who furnished Dr. Martin with the following observations were under the care of Dr. Déjèrine, at the Bicêtre; out of 106 cases of tabes dorsalis he found 21 who were amaurotic, and who in addition to optic atrophy suffered from lightning pains, loss of knee-jerk, and other unequivocal symptoms of the disease. A full account is given of the clinical history, and present state of each of the cases, and the most important points are brought out in the following analysis.

The cases fall into two groups; the first group comprising Nos. 1-15 inclusive, are in the pre-ataxic stage, or in other words during a greater or less number of years have developed no ataxy of movement. The duration of this pre-ataxic period has been from three to thirty-three years in different cases, and in ten instances varies from ten to thirty years. In the second group of six patients optic atrophy came on as a secondary phenomenon to pre-existing ataxy.

Taking the first group, and leaving on one side the loss of the knee-jerk on account of the impossibility of determining the date of its disappearance, the first sign of the onset of the disease in nine cases was the occurrence of lightning pains with or without concomitant amaurosis; in six patients it was amblyopia with or without oculo-motor troubles, and in three of the latter lightning pains only came on seventeen, eighteen, and nineteen years respectively after the development of complete amaurosis. Of eleven patients who suffered from lightning pains at the time when amaurosis was fully established, in five the pains have been ameliorated since the loss of sight, in six they have remained stationary, and in no case has an exacerbation of the pains occurred.

In two cases who show ataxy of movement subsequent loss of sight has been followed by relief of pain.

In the second group, where amaurosis supervened on ataxic symptoms, the steady progress of the disease has been unchanged. In one case only ataxy came on six years after total loss of sight, and in this patient as in the others blindness was accompanied by amelioration of pain. The degree of ataxy was slight, and another exceptional feature was the presence of Romberg's symptom, which is not generally found in blind tabetics.

Benedikt has stated that besides atrophy of the optic nerve, the appearance of other symptoms, notably gastric crises, modifies favourably the course of the disease. This observation the author has been unable to confirm; in the cases he has observed, the appearance of gastric crises has had no influence on the further progress of the spinal lesion. As to the question of the influence of syphilis, in only thirty-three per cent. of his patients was there a syphilitic history. This percentage is below that generally given for all cases of tabes dorsalis, so that syphilis does not seem especially to lead to optic nerve atrophy. He finds also that patients with oculo-motor paralyses do not suffer more frequently from amaurosis than those in which these paralyses do not occur. He concludes that these cases of "abortive" tabes with optic nerve atrophy are not to be arbitrarily separated from the more typical cases; their chief peculiarity lies in the rudimentary state of development of the spinal symptoms, in the common amelioration of the pains, and in the extreme rarity of the occurrence of locomotor ataxy. Caution must be exercised in attempting to find a single explanation of the different manifestations of tabes, and all the more as the present tendency is to decentralization in the pathological anatomy of this affection. The discovery of neuritis of the sensory nerves singularly alters our conceptions of the pathology of tabes. As Déjèrine observed in 1882, the spinal lesions being equal, the motor and sensory troubles may show great variation from one case to another, and this variability in the symptoms chiefly depends on the greater or less extent to which peripheral neuritis occurs. The disease can no longer be considered simply as an affection of the spinal cord, but rather as a malady in which, together with fundamental spinal changes, the nerves of common sensation, of motion, and of the special senses always present changes of variable extent.

On the Retention of the Patellar-Tendon-Reflex in Cervical Tabes Dorsalis. By Professor Eichhorst. Beiträge zu Pathologie der Nerven and Mustreln. With a Plate. Fortsetzung von Bd. 120, S. 217.

Westphal by his researches, which stand as an example of what may be accomplished by accurate clinical observation, proved that in diseases of the spinal cord the patellar reflex is absent when one particular spot is damaged, and that this spot is situated at the entrance of the posterior roots (or internal root zone), from the lower dorsal to the upper lumbar portions of the cord; moreover where the patella reflex is lost on one side only it is lost on the same side as that on which the disease of the cord exists.

Many other observers have since amply confirmed his conclusions. When the knee-jerk disappears early in tabes dorsalis, the most advanced anatomical changes will be found in the lower part of the cord, and first arise in the position above mentioned. It is however conceivable, and Westphal's latest observations afford some examples, that the morbid lesion in tabes may begin exceptionally in the lowest segments of the cord, and leave the above described portion undamaged, or may affect first the cervical region, and only gradually spread to the parts of the cord below. In the latter case the patellar reflex will be retained so long as the pathological alterations remain above the lower dorsal and upper lumbar regions, and do not affect the posterior internal root-zone in this situation.

Weir Mitchell has reported a case in which ataxic symptoms began in the arms; the tendon reflexes were lost in them whilst the patellar-reflexes were exaggerated. He takes occasion to remark that the disappearance of the reflexes in tabes is preceded by a stage of exaltation. It is to be regretted that there was no anatomical examination of the cord in this case. Martius describes a case in which the patellar reflex was present during life, and the subsequent examination of the cord showed that the degeneration was most extensive in the cervical region, and that the posterior internal root-zone in the lower dorsal and upper lumbar regions was not damaged.

Professor Eichhorst then describes his case which bears on the relation between the patellar reflex and tabetic changes in the cervical region of the cord. The patient, a woman of fifty-four years of age, was admitted in a comatose state suffering from an apoplectic seizure which had caused right hemiplegia. From the patient's condition no information as to her past history could be obtained from her, and she was a stranger to the people with whom she was living when struck down by the illness. The left ventricle was dilated and hypertrophied and the urine, withdrawn by catheter, contained a trace of albumen. She was a big woman, but her muscles were small and flabby, especially those of the legs. Moreover, the knee-jerk was absent on both sides. This fact made it likely that the patient was suffering from hemiplegia of cerebral origin coming on in the course of tabes dorsalis. She died in the evening of the third day after the onset, her temperature being 101·5°, and 103° half an hour after death.

At the autopsy a large hæmorrhage into the white substance of the left hemisphere was found, which had ruptured into the lateral ventricle. The left ventricle of the heart was hypertrophied, on the mitral valve were a few thickenings of old date, and in the kidneys the surface of the cortex was marked with numerous scar-like depressions, and their capsules stripped with difficulty.

With the exception of some smooth, whitish patches, not calcareous, in the arachnoid the spinal membranes were healthy; the cord was hardened in Müller's fluid, and stained by Weigert's and other methods. Contrary to expectation the lower dorsal and the whole lumbar regions were normal in all aspects. In the mid-dorsal region the posterior columns were almost completely degenerated, the morbid process being most advanced in the posterior root-zone. Briefly the ordinary lesions of tabes dorsalis were found in the cervical region, and in the dorsal as far as the junction of its middle and lower thirds. Good coloured drawings are given of the morbid appearances. Careful microscopic examination showed that the stress of the disease had fallen upon the mid-dorsal region in which, together with the almost complete absence of medullated fibres in the posterior columns, the cells and fibres of Clarke's column had also degenerated, and that the lower third of the dorsal and whole lumbar regions were unaffected by the disease. The microscopic appearances made it probable that the lesions had existed for some time.

Here then, was a case of tabes, in which although the knee-jerks were absent the cord was healthy from below the mid-dorsal region to the filum terminale. If the investigation had stopped here an erroneous conclusion might have been arrived at. Further examination, however, showed that the anterior crural nerves had been the seat of neuritis; sections of these nerves proving that very few medullated fibres remained, and these lay for the most part in the periphery of the nerve under the perineurium. The loss of the patellar reflex is, therefore, to be attributed in the healthy condition of the lower part of the cord to this neuritis of the anterior crural nerves. The author concludes by saying that in ordinary typical cases of tabes the cause of the loss of the patellar reflex is to be sought in the changes in the spinal cord, for the patellar reflex is generally lost in the earliest stage of the disease, whilst neuritis of the peripheral nerves occurs in the late periods, but he wishes to emphasize the fact that in rare cases of the disorder the cord may be normal in the lower dorsal and upper lumbar regions, while the patellar reflex is absent, and in these cases the loss of the knee-jerk may be due to changes in the peripheral nerves causing an interruption of the reflex arc.

A Case of Locomotor Ataxia associated with nuclear Cranial Nerve Palsies and with Muscular Atrophies. By FRED. PETERSON, M.D. (from *Journal of Nervous and Mental Disease*, 1890).

The patient was under the care of Dr. Seguin, from 1884-1886, and came under Dr. Peterson's observation in March, 1890.

M. J. T., æt. 37, had syphilis in 1875. In 1882 he had diplopia, dimness of vision and lightning pains; In 1883, a momentary loss of consciousness in which he fell cutting his head. He had also syphilitic disease of the testicle. In 1884 there was paralysis of internal rectus, inferior oblique, and sphincter iridis, and paresis of the superior and inferior recti in the right eye. In the left eye the muscles supplied by the third nerve acted variably and feebly. The pupils were inactive to light and to accommodation, and the left pupil was larger than the right. No lesion of optic nerves. Slight paresis of right hand and left side of face; knee-jerks exaggerated; micturition both feeble and involuntary. In 1885, partial right, and nearly total left, ptosis. Condition of muscles in right eye unchanged, in left much improved. Some paresis and atrophy of temporal and masseter muscles on each side. In 1886, the knee-jerks previously exaggerated had fallen to normal. Dr. Sequin writing in 1888 (*Journal of Nervous and Mental Disease*, May, 1888), said, "The fulgurating pains with hyperalgesia, and the fall in degree of the knee-jerk during two years of observation would seem to justify a condition of incipient posterior spinal sclerosis."

Present condition; almost complete ophthalmoplegia externa and interna, except that in the right eye the external rectus acts, but is paretic, and in the left the superior and inferior oblique muscles move a very little, and very feebly. Pupils widely dilated, equal and immobile. Accommodation paralysed. Vision R.V. $\frac{20}{70} + \frac{1}{18} = \frac{20}{40}$. L.V. $\frac{20}{50} + \frac{1}{18} = \frac{20}{40}$.

Weakness and atrophy of the temporal and masseter muscles if anything more pronounced. The pterygoids on each side are also paretic. The left side of the face is a little paretic, and the tongue deviates slightly towards the left. Other cranial nerves normal. There is no anæsthesia of the face. The right-hand is weak. There are loss of knee-jerks, great ataxia of all four extremities, inability to walk without assistance or to pick up a pin. Numbness, analgesia and anæsthesia over fingers, and pronounced loss to all kinds of sensation over the lower extremities, and on the trunk as far as the umbilicus. He has incontinence of urine, and weakness of anal sphincter. Plantar and cremasteric reflexes absent. He has never had any crises. In the left upper extremity there is complete paralysis of the two long extensors of the thumb, and atrophy and paresis with reaction of degeneration of the abductor minimidigiti, interossei and lumbricales. In the right-hand the opponens and abductor pollicis show almost complete atrophy. The adductors of both thighs are considerably wasted, and the right thigh is smaller than the left, while the left leg is smaller than the right. Degeneration reactions were present in the atrophied muscles.

The author's diagnosis of the case is indicated by the title of the paper. He thinks that the solution of the question whether the muscular atrophies are due to peripheral or central lesions must be left to the autopsy. The ophthalmoplegia he considers to be nuclear. Although it has been assumed by a number of authors that total paralysis of all the muscles supplied by the third nerve implies not a nuclear, but a nerve trunk palsy, in this case it would be, to say the least, difficult to conceive of a lesion at the base of the brain so widely, and so symmetrically distributed as to affect the trunks of both third, both fourth, and both sixth nerves, and the motor portions of both trigemini, and yet to permit the escape of the sensory portions of the latter.

The forms of Tabes in Infancy. By Prof. A. Pick. (*Sep. aus der Zeitschr. für Heilkunde*, xii., 1891.)

Since Kahler and Pick first expressed the opinion that the origin of the combined system scleroses was to be sought for in developmental defects of the systems of fibres involved, this hypothesis, although at the time pretty generally accepted has made little further progress. Professor Pick has further reported a case in which he discovered in the cord an anomaly approaching to an absence of development of the pyramidal tracts, and has indicated the points of similarity between this case and two cases in which there was a combined system sclerosis. After ten years he is still of opinion that the solution of the question lies in the advancement of the demonstration of this hypothesis, and in the further investigation of it in this paper he considers—(1) cases of idiocy in which, besides the developmental defects of the brain, there are similar defects in the cord, which are presumably of the nature of a "Hypoplasia" of the "systems" affected. (2) Those cases in which, as in Friedreich's disease, the development is different, but there is a clinical correspondence in the spinal symptoms.

He begins by reporting fully a case which seems to form a connecting link between the two preceding groups.

The case is that of a young man age twenty-one, imbecile, and paralysed from infancy, and lately restless, mischievous, and destructive. His father and mother were healthy, but the latter had a severe fright in the fifth month of her pregnancy. The patient was weakly from his birth, his head was large, and from the age of a few months showed nodding movements. He could neither sit nor stand. Chorea was diagnosed at the age of three; he never was able to walk properly; his arms were strong, but at the age of fifteen already showed tremor. Until four years old he was fairly well nourished, but at the age of eleven he had an attack of erysipelas, and since then he has never been so well. Mental development very backward; he speaks very badly, stammering, and repeating words. He showed, however, considerable aptitude for wood carving. On admission, the patient was a little over five feet in height, pale and emaciated. He lay in bed with the knees flexed, and the head bowed forward so that the chin rested on the sternum. Forehead low and broad; frontal and parietal eminences prominent; the head held a little to the left oscillated with swaying movements which corresponded to similar movements of the trunk. The mouth was constantly opened and shut, with movements of the lips and tongue as in swallowing. The eyes were perpetually affected with rapid movements chiefly in a horizontal direction; no nystagmus; pupils acted well. From spasm of the eyelids the eyes were always kept half shut; so far as could be ascertained vision was normal. Optic discs pale, but the retinal vessels of normal size. The limbs were agitated by irregular inco-ordinate movements, which were increased on voluntary motions, and interfered with the due performance of them. The arms shared in the motor disturbances, and an attempt to lift the arm resulted in movements at the wrist-joint, and of flexion and extension of the fingers by which the limb was shifted along the bed. The muscles of the upper arm, especially the pectorals and deltoid, were feebly developed, those of the fore-arm were well formed. The legs were wasted when he lay in the dorsal position, the left being rotated outwards and flexed at the knee. The toes were turned towards the sole, and the feet were in the position of talipes equino-varus. Electrical re-actions normal. Scoliosis of lumbar spine with convexity towards the left. Superficial reflexes active; knee-jerks absent. The patient without support could only stand for a second, and after that had to be supported on both sides. On trying to walk he raises the left leg stiffly, flings it in front of him, without moving forwards the body, which tends to fall backwards. He could not balance himself. Sensation was unaffected.

In the further course of the disease the choreiform movements became less marked, the ataxic correspondingly more prominent.

The motor unrest diminished gradually, and the patient often lay quite quietly. He learnt a few words; speech was monotonous and nasal; the words being uttered in an explosive manner, and being for the most part incomprehensible. His intelligence remained at the same level, and he was easily irritated.

Professor Pick then proceeds on the basis of the present state of knowledge of neuro-pathology to give, first of all, an explanation of the fore-going case without reference to any similar form of disease. The positive symptoms available for this purpose are the congenital mental feebleness, the general paresis of the voluntary muscles, the inco-ordinate movements, and the loss of the knee-jerk, whilst a number of other symptoms are not capable of receiving at present an anatomical explanation. As regards the idiocy, that is to be attributed to congenital hydrocephalus, and this view is not contra-indicated by the fact that the circumference of the skull in not greater than normal, for chronic internal hydrocephalus may co-exist with a relatively small skull.

The paresis of the voluntary muscles present appears to warrant the conclusion that whether primarily or secondarily, the direct paths for the passage of motor impulses, the pyramidal tracts, are in a condition of impaired function, which if we bring in the genetic factor, may be expressed in pathologico-anatomical terms, as a want of development or "hypoplasia" of these columns.

The inco-ordination of movement, and absence of the knee-jerk taken together show an abnormal state of the posterior columns, and, when we consider the history of the case, this abnormal state may be referred to the fœtal development, and spoken of as an "hypoplasia," or at least a condition predisposing to the later disease of these columns. Here it is to be noted, that the ataxic symptoms had not altogether a clearly tabetic character.

Apart from the brain disease we could postulate for the cord a degeneration of the lateral columns extending throughout the medulla spinalis, and of the posterior columns in the systems of fibres above-defined, extending into the lumbar region of the cord. The presence of this disease of the posterior columns would explain the absence of spastic symptoms.

So far as concerns the affection of the pyramidal tracts it is important to note that the action of the clearly specified ætiological factor took place at about the time at which the development of the pyramidal tracts occurs. Without entering into the discussion of cases of hypoplasia or absence of development of the pyramidal tracts in which the lesion has been erroneously referred to a secondary degeneration arising from defects of cerebral development, Professor Pick mentions cases in which congenital hypoplasia has been found in the pyramidal tracts (Anton), posterior columns (Leyden), pyramidal tracts and columns of Goll (Hervouet and also Flesch-Steinlechner), and points out that many of the symptoms in these cases correspond with those present in his own. Exact observations on the motor functions in idiots are wanting, but the author quotes as worthy of remark in connection with the preceding observation some symptoms described by Schüle (*Klinische Psychiatrie* 3 *Aufl.*, *S.* 509), which closely correspond with those present in the case under discussion.

Concluding from the preceding data that in the case of his patient there is a combined hypoplasia of the lateral and posterior columns of the cord, the author then passes on to compare it with Friedreich's hereditary ataxia, in which such a hypoplasia has been considered to be the ground-work of the disease. In Friedreich's ataxia the spinal symptoms ought, *a priori*, to stand out in clearer relief as they are not disguised by developmental brain defects, and without being able to claim a correspondence or analogy for the foregoing case with Friedreich's disease on the results of an anatomico-pathological investigation, there are many factors, apart from the similarity of the clinical symptoms, which show the close connection between them.

It is not uncommon for hereditary ataxy to be developed in the first childhood, at a time when the development of the pyramidal tracts is just completed, and for the first ataxic symptoms to appear when the children are learning to walk, so that in this respect there is a likeness between the two. Professor Pick believes that the pathogenic factors which produce the differences between the two diseases do not come into play so far as the spinal cord is concerned, and it can be proved that this also holds good for those factors which are sufficient to explain these differences.

First as to the time of onset of the hypoplasia. There is a difference according as the affected columns are first implicated, either at a time when the development of the most important parts of the brain is also proceeding, or on the other hand at a time when they together with the brain have made essential progress in development, in which case there is perhaps only an arrest of formation of the medullary sheaths. Then with regard to the extent and intensity of this arrest; in Friedreich's disease we have to deal with further evolution of the morbid changes, in the other cases now under consideration with stationary symptoms and lesions.

The next step in the enquiry is to ascertain how far the symptoms in this case correspond with those of Friedreich's ataxia, and in this direction Professor Pick found that his expectations were far surpassed. He gives a summary of the leading symptoms from Ladame's account of Friedreich's disease (*See* BRAIN, *Winter Number*, 1890). In the following symptoms his case corresponded with hereditary ataxia. Chorea, which is a characteristic of the first stage in those cases where it exists in addition to the other symptoms of Friedreich's ataxia. Though exact information was wanting, the progress of the interference with movement in his case was probably slow, and gradually increased; similar disorders of the oculo-motor muscles, and involuntary movements epecially in the legs have been found in particular cases of hereditary ataxia. The partly ataxic, partly staggering (cerebellar) character of the gait has been also described by Charcot and Soca; the absence of any remissions in the course of the disease, the movements of the head, the loss of the knee-jerk, and the talipes equinus are characteristic, while the "explosive" character of the articulation mentioned above, though not conforming to the typical disorder of speech described by Ladame, has been noted in other cases by Friedreich as "ataxy of speech," and by Griffith as jerking articulation. Lastly, nystagmus has been absent in undoubted cases of Friedreich's disease. As regards the differential diagnosis the case could not be considered to belong to any variety of chorea, to tabes dorsalis developing at a very early age, to disseminated sclerosis, nor to any of the known forms of combined "system" diseases of the spinal cord. To sum up, it is a case of idiocy in which the disorders of movement are to be attributed to defect of development of the spinal conducting systems, and which, on account of the similarity of the clinical symptoms, stands in close relationship to Friedreich's disease; the clinical points of difference being, perhaps, explained apart from the affection of the brain, sometimes by variations in the time of onset of the cause giving rise to the hypoplasia, sometimes by the same cause producing a different local effect.

With these considerations, and the diagnosis based upon them the autopsy was made in April, 1890, the day after the death of the patient from tuberculosis.

The skull-cap was found to be thick and remarkably compact; together with the meninges, the right and left hemispheres each weighed 550 grammes; the cerebellum, medulla and pons weighed 180 grammes. The cranial nerves were normal. Membranes not adherent; convolutions appeared normal in arrangement and configuration; the third ventricle, and the aqueduct of Sylvius were dilated in size, and the ependyma thickened and condensed. Sections through the cerebrum and cerebellum showed no pathological alterations.

Spinal cord. Meninges soft; the cord was flattened over the posterior columns; in the cervical region Goll's column was grey in colour as was also the middle of Burdach's column. The cord was pretty firm in consistence, but was in all parts smaller than normal.

In the larynx were two ulcers exposing the perichondrium of the left arytenoid catilage. Tonsils enlarged. Extensive tubercular lesions were found throughout the lungs. The pleuræ contained serous effusion. The kidneys also showed morbid changes.

Microscopical examination showed degeneration of Goll's and of Burdach's columns, which must most probably be regarded as system-lesions; the direct lateral cerebellar tract was similarly affected, but this could not with certainty be considered as a system degeneration. The pyramidal tracts, and the grey substance of the cord were unaffected. This microscopic examination must be taken as provisional only, as the hardening of the cord was not completed.

Professor Pick concludes that besides typical tabes dorsalis occurring in childhood, and in close relation with the characteristic form of juvenile tabes described by Oppenheim, due to a combined affection of the posterior and lateral columns, including the pyramidal tracts, there is a symptom-complex associated with imbecility which resembles Friedreich's disease, and depends on degeneration of Goll's and of Burdach's column together with the direct lateral cerebellar tract whilst the pyramidal tracts are unaffected.

Flaccid Paraplegia from Compression of the Cord. By J. Babinski.

The author begins by defining spastic paraplegia as a disease characterised by rigidity of muscles, and contraction of the lower extremities with exaggeration of the tendon reflexes and ankle-clonus, and flaccid paraplegia as distinguished from it by the relaxed condition of the muscles, flaccid state of the limbs, without exaggeration of the tendon reflexes, generally indeed, with diminution or abolition of them. He then discusses the physiology of the tendon reflexes, and in the case of the knee-jerk considers that there must be a reflex-centre seated in the lower part of the dorsal region of the cord and in the upper part of the lumbar enlargement. The conditions in which the tendon reflexes are exaggerated, and diminished or abolished are enumerated. Special reference is made to the observations of Bastian and Bowlby, who have shown that in a case of the lesion occupying the whole transverse extent of the cord in the lower cervical or upper dorsal regions, and followed by a descending sclerosis of the pyramidal tracts there is abolition of the tendon-reflexes, and paralysis of sensation and motion below the lesion together with disorder of micturition and defæcation. If, on the other hand, the destruction of the cord is incomplete at the seat of the lesion the tendon-reflexes are exaggerated.

Two cases are then reported, the first that of a man aged 64, who had suffered for six weeks from complete motor paraplegia without muscular atrophy, and without anæsthesia, but with vesico-rectal troubles, and a bedsore over the sacrum. The muscles were flaccid, and the tendon reflexes completely abolished. Microscopic examination showed that the cord in the regions compressed presented signs of slight damage only, and that the secondary degeneration of the pyramidal tracts was altogether rudimentary. Death resulted from amyloid degeneration of the kidney and pyelo-nephritis. The second patient, a man aged 71, had been subject for more than a year to transitory attacks of vertigo accompanied by weakness of the legs. One day after an attack of giddiness he fell down, and could not get up again; he was taken simultaneously with paraplegia, and with right hemiplegia affecting the arm and leg, but not the face. The paralysis of the right arm soon disappeared, the paraplegia, however, remained, it was not quite complete, but the patient could only accomplish a very limited degree of movement. At the same time there were rectal and bladder troubles, and a bedsore over the sacrum. There was no atrophy of muscles nor loss of sensation, the muscles were flaccid, and the tendon reflexes diminished. The paraplegia persisted for five months until the patient died apparently of septicæmia. Microscopical examination showed that the spinal cord was normal.

The author quotes two other cases of the same kind, published by Kadner and by Druschky, which are, however, very meagrely reported, and says, that these observations show that compression of the cord in the cervical or dorsal region can, without giving rise to any appreciable organic lesion, produce a motor paraplegia lasting several months, accompanied by vesico-rectal troubles and bedsores. He concludes provisionally that when flaccid paraplegia has lasted more than a month, and there is no lesion of the reflex arc, nor destruction of any part of the cord throughout its thickness, the nerve fibres of the cord are intact, or at least very little damaged. An exact diagnosis may be made clinically. In the case of a patient, for instance, suffering from flaccid paraplegia caused by morbid changes in the upper or middle dorsal vertebræ, in whom the paraplegia has existed for several weeks, and there are no signs of damage to any of the segments of the nervous arc of the tendon reflexes, we have to determine whether the paraplegia exists without organic lesion as in the two cases above reported, or is caused by irreparable damage to the cord, as in the cases observed by Bastian. The distinction is made by the presence of complete anæsthesia in the latter, and is important from the point of view of prognosis and treatment. In the first group recovery is possible, and the author thinks that trephining of the spinal column with the removal of the tissues compressing the cord would be here particularly advantageous. He draws the following conclusions :—

(1.) Compression of the cord can, without giving rise to appreciable organic lesion, cause very severe and even complete paraplegia of several months duration.

(2.) When, in flaccid paraplegia which has lasted some weeks and is consecutive to compression of the cord, the flaccidity cannot be attributed to a lesion in any part of the reflex arc of the tendon reflexes, and is, moreover, not due to the destruction of a portion of the cord thoughout its entire thickness, we may conclude that the cord is not altered or only very slightly damaged.

(3.) In this variety of paraplegia, other things being equal, the prognosis is relatively favourable.

(4.) Surgical interference is especially indicated if the general condition of the patient does not contra-indicate it.

J. Michell Clarke.

Kurella on Asymmetry of the Skull in Torticollis.

Centralbl. f. Nervenheilk u. Psych. August, 1891.

The mechanical factors which condition the external conformation of the skull are but imperfectly known. In considering the causation of deformities of the head, the attention has been chiefly directed to those circumstances affecting the degree of cranial resistance to the expansive pressure of the skull contents, and a whole series of cranial malformations has been explained by variations in the time at which ossification of sutures has occurred in symmetrically placed regions.

The influence of the traction force exercised by the deep muscles uniting the spinal column to the occiput, in cases of scoliosis, has been treated in an able work on the "Scoliotic Cranium," by L. Meyer, but the nearly allied deformities of the skull in torticollis have not previously been specially described.

To render this gap in literature less complete, Dr. Kurella relates the case of a village cobbler, aged forty-six, who came under observation owing to his criminal propensities. The patient was brutal, intemperate and somewhat imbecile ; he exhibited a slight degree of *caput obstipum* with very considerable asymmetry of the skull. Patient attributed his torticollis to a fall against the edge of a table during his first year. For nine years after the accident he carried his head obliquely backwards and to the left, with his chin turned to the right. About his tenth year this condition began to lessen. From his thirtieth year only the slight deviation, noted above, remained, and there was no impediment to the normal voluntary movements of the head. Dr. Kurella found the portion of the right trapezius arising from the occipital bone greatly atrophied; no marked difference between the right splenius capitis and the left was evident; the right sterno-mastoid, particularly its sternal portion, was much thinner than the left. Forehead, very receding; presented a deep groove above the enormously developed superciliary ridges; left half of forehead small and less vaulted than the right; left frontal eminence scarcely discernible; right, strongly developed. Subnasal portion of face prognathous. Left side of hard palate much the wider and less arched. Left parietal eminence more prominent, and situated more posteriorly, than its fellow. Left mastoid process very strongly developed, while the right was barely perceptible. The left external meatus situated nearly two cm. lower than the right. Left occipital region considerably the wider and more projecting.

Cranioscopically the skull appeared as though compressive force had acted on the left side anteriorly and the right side posteriorly, the left half being at the same time drawn downwards in the petrous and mastoid regions, and bulged downwards and outwards in the occipital region.

The length of skull was 180 mm., breadth 158, the skull accordingly was brachycephalic, with an index of 87.7. The smallest width of forehead equalled 100 mm. The frontal index was strikingly great—surpassing that described by Corre (viz., 0.71) as considerable for a murderer's skull. The distance from the occipital protuberance to the left frontal eminence equalled 180; to the right, 183 mm. Horizontal circumference 545 mm., of which the left half barely took 270, a difference which could not be considered to rest exclusively upon errors of measurement. An accurate estimate of asymmetry by linear and arc measurements during life, however, is scarcely possible, or at best the craniometric procedure gives questionable lustres.

A contour delineation of the normal vertical gave a figure very similar to that of the skull designated scoliotic by Meyer.

Dr. Kurella indicates how the cranial deformity resulted from the prolonged tonic spasm of the left sterno-mastoid, trapezius, and splenius capitis, the most prominent evidence of the abnormal muscular action being the lowered position of the external meatus and the marked downward bulging of the occipital bone. The palatal and maxillary deformities must also be considered in relation with the permanent (during nine years) unequal pressure on the condyles from the traction on the basis cranii.

The left half of the skull proved, on the whole, to be less developed than the right, hence arises the question whether the constant pressure on the vessels, particularly the carotids, of the side affected with spasm may not be educed in explanation of that inhibited general growth, as Bardeleben has done for the scoliotic skull? In this case of Kurella's that assumption seems to gain in probability from the fact that considerable asymmetry of the larynx co-existed; the left half of the thyroid cartilage stood almost in the middle of the neck, on a lower plane than the right, and formed with the latter an angle nearly aproaching 90°.

Dr. Kurella does not enter into the connection between the mental anomalies and the cranial deformity further than to remark upon the frequent occurrence of malformation in the occipital bone in habitual criminals.

Schütz on the Nerve Fibres of the Central Cavity—Grey Matter and Their Degeneration in General Paralysis of the Insane. *Arch. f. Psych. u. Nervenkr.* Bd. xxii. H. 3.

With the aim of throwing some light upon the pathologico-anatomical basis of reflex pupillary inaction in general paralysis—a symptom which occurs in about 50 per cent. of cases, and may be considered to be one of the early indications of this disease—Dr. Schütz examined first the peripheral portions of the reflex arc. The results were negative; in 20 cases of general paralysis with reflex pupil immobility, the retina, papilla, optic nerve, chiasma, third nerve, and ciliary ganglion exhibited no pathological change. These cases, during life, had presented normal ophthalmoscopic appearances.

Elaborate researches into the normal histology of the central grey substance in the adult, infantile, and fœtal brain, were then necessitated by the facts that, although the cellular elements of the nerve nuclei have been studied and described by many investigators, comparatively little has been written upon the fibres of the central grey, and not until Pal's method of staining came into use has it been possible to trace, or even see, many of the delicate medullated fibres which form part of its extremely complex structure.

These researches showed that from the tuber cinereum to the pyramidal decussation three fairly defined formations can be distinguished in the central grey matter of adults, viz., (1) the nuclear grey, (2) the retiform grey, and (3) the system of longitudinal fibres (dorsal longitudinal bundle).

In the first the author includes Stilling's nuclei, the nucleus funiculi teretis, Westphal's trochlear nucleus, and those groups of ganglion cells in the central grey which are not regarded as centres of origin for root-fibres of cranial nerves.

The retiform grey is most abundant in the region of the aqueduct of Sylvius. Near the aqueduct it receives fibres which radiate from the quadrigeminal bodies and probably are connected with the oculomotor nucleus, also a bundle of fibres, apparently proceeding from the red nucleus, whose further course could not be traced.

Many of the fibres running in the central grey matter arise from the thalamus. Two tracts emerge from this body; one from the chief nucleus and ganglion habenulæ, the other from the antero-ventral portion of the thalamus. They enter the central grey somewhat in front of the middle commissure and course towards the mouth of the aqueduct; here they meet a fasciculus from the tuber cinereum which derives its fibres partly from the network of the latter and partly from the nucleus of Luys and the ansa peduncularis. After passing the mouth of the iter the fibres of the three bundles radiate in various directions; some, probably coming from the chief nucleus, enter the posterior commissure and mingle with its fibres; another portion traverses the roof of the aqueduct to end in the corpora quadrigemina and reticular grey substance. Apparently, other fibres, best seen in sagittal sections, run immediately beneath the ganglion of the posterior corpus quadrigeminum through the anterior medullary velum into the cerebellum. But the chief mass of fibres in the central grey, directed caudalwards from the third ventricle, constitutes the dorsal longitudinal bundle. This lies along the floor of the aqueduct, between the lining and the oculomotor nucleus, spreads into a thin layer immediately beneath the ependyma of the fourth ventricle, as far as the vagus nucleus, then is collected into a club-shaped mass in the region of the hypoglossal nucleus, and continues downwards to the central canal.

The dorsal longitudinal bundle gives fibres to all the cranial nerves, to the previously mentioned cell groups—*e.g.*, Westphal's trochlearis nucleus and the superior central nucleus, to the retiform grey, and to the formatio reticularis grisea, as well as to parts external to the central grey.

The fibre-network of the several nuclei varies much in closeness; the hypoglossal nucleus has the richest plexus of fibres; next come the oculomotor, facial and remaining motor nerve nuclei. As in the cord, the network of the sensory nuclei is much less close than that of the motor.

The fibres of the central grey matter have no medullary sheath until after myelination of most of the other fibre systems. In children, newly-born or a few weeks old, the dorsal longitudinal bundle, and the reticulum of delicate fibres in the nuclei of nearly all the cranial nerves are not visible; the root fibres alone are medullated: the radial fibres entering the formatio reticularis grisea and those from the ansa peduncularis and nucleus of Luys also are non-apparent. The oculomotor and trochlearis nuclei develop their network about the seventh month of fœtal life.

Dr. Schütz acknowledges that his researches, the description and results of which are very fully communicated in his paper, leave many voids—"The chaotic intricacy of the fibres in various regions of the central grey often makes all attempts to recognize any definite arrangement of them completely illusory."

The clinical and pathological conditions found in twelve cases of general paralysis are detailed. For the purpose of this abstract we give an epitome of Case II., selected at random:—

A female general paralytic with paresis of right elevator of eyelid and internal rectus. Pupils of medium size, right pupil larger than its fellow, both inactive to light, tongue tremulous, articulation impaired, knees' jerk absent. Later there were right facial paresis, Romberg's symptom, and marked ataxy.

Post mortem:—Posterior spinal sclerosis in upper part of cord; nuclei of posterior columns shew well-preserved fibre-net-work.

The dorsal longitudinal bundle greatly atrophied from the decussation of pyramids to the oculomotor nucleus; thence the degeneration gradually diminishes, but is still evident at the level of the posterior commissure. Over the hypoglossal nucleus the fibres of the dorsal longitudinal bundle shew numerous varicosities. From the auditory nucleus to the locus cæruleus there are many small blood vessels, with thickened walls, amongst the fibres of this tract.

Few fibres of the retiform grey in the neighbourhood of the aqueduct remain.

In the hypoglossal nucleus the root-fibres appear sharply defined amidst the mere vestige of the fibre-network. The facial and vagus nuclei, the nucleus funiculi teretis, and the superior central nucleus exhibit a somewhat minor degree of this atrophy. The reticulum in the nuclei of theother cranial nerves is not affected.

In the vicinity of the locus cæruleus and the descending root of trigeminus, the fibres passing into the formatio reticularis from the central grey, are greatly reduced in number. The radial fibres from the deep medullary layer of the anterior quadrigeminal body to the central grey are remarkably few, especially the lateral group, as compared with the normal condition.

In all the cases there was more or less pronounced atrophy of fibres in the dorsal longitudinal bundle. In some cases this atrophy extended from the upper limit of the pyramidal crossing to the third ventricle ; in other cases it ceased at the proximal end of the aqueduct. With one exception, all the cases shewed a decrease of fibres in the ventricle from the region of the locus cæruleus to that of the oculomotor nucleus, the diminution being most marked in this latter area.

Concurrently with the degeneration of the dorsal longitudinal bundle, there was decrease of the fibres radiating into the formatio reticularis from the central grey, and the retiform grey was affected in equal degree.

The nuclear grey, in all but one case, displayed marked atrophy of its fibre-network.

Of the cranial nerve nuclei only the motor were involved. The hypoglossal nucleus was most degenerated in nine of the cases; in some of these the atrophy of the plexus had proceeded so far that only the root fibres remained. The fibre-network of the facial nucleus was greatly diminished in eight cases. The nuclei of the other cranial nerves were mostly unimplicated in the fibre degeneration.

The nucleus funiculi teretis in two cases was almost free from fibres. Usually the superior central nucleus shewed atrophy of its retiform structure.

In most of the cases there was no trace of inflammatory process in the central grey substance. About half of the cases shewed, in places, a considerable number of newly-formed vessels with thick walls, but without proliferation of nuclei around.

Several considerations lead Dr. Schütz to think that the fibre degeneration in the central grey is a primary systemic affection, in its restriction analogous to systemic lesions in the cord. He maintains that the fibres of the central grey matter constitute a distinct system, a fact which he considers is proved by developmental circumstances—the late myelination and small calibre of the fibres, and their apparent absence until some weeks after birth at term. The fibre-atrophy is confined to those fibres whose developmental history is specifically distinct from other fibre systems. A section from the brain-stem, or medulla oblongata of a general paralytic, with extensive fibre-atrophy in the central grey, presents a similar appearance, in this respect, to a section from the same part of a newly-born child; in both the dorsal longitudinal bundle, the network in the motor nerve nuclei (excepting the oculomotor), and the fibres streaming into the formatio reticularis are absent.

Dr. Schütz does not assume to have established that the degenerative condition described by him is a lesion peculiar to general paralysis, or that it occurs in all cases of that disease. In senile dementia, chronic alcoholism, and paranoia he has found no such condition.

Which of the symptoms of general paralysis are to be assigned to the degeneration of fibres in the central grey? Dr. Schütz admits that the present state of our knowledge is inadequate to solution of the question. In general paralysis nearly all parts, peripheral or central, of the nervous system have been found diseased. Most recently the researches on degeneration of fibres in the cerebellar cortex, and those of Lissauer on disease of the thalamus in general paralysis have rendered the attempt to discover the relationship between the semeiology and pathology yet more difficult. Further, the physiology of the central grey matter is almost unknown. Dr. Schütz thinks that the network of the nerve nuclei represents a system of association fibres between the individual cells of the nuclei, by which is conditioned a regulated synergy of the ganglion cells. In like manner the dorsal longitudinal bundle would have a similar function for groups of ganglion cells. The fibres of the central grey matter accordingly serve functions which do not come into existence until some time after birth, and which are expressed by co-ordinated action of groups of muscles in complicated fine movements, such as articulation. It is precisely these fine movements which are the first to become impaired in general paralysis.

The infrequency of sensory disturbances and derangement of the external eye-muscles in general paralysis corresponds to the results found by Dr. Schütz, viz., absence of evident change in the oculomotor and sensory nuclei of the caudex. He concludes that disease of the cerebral cortex can no longer be regarded as the most essential cause of the motor derangements in general paralysis. In his cases, with one exception, the severity of the motor symptoms coincided with the intensity of the fibre-degeneration in the central grey.

He confesses that he has failed in the primary object of his research.

ERNEST BIRT.

Original Articles.

ADDENDUM TO Dr. WALLER'S PAPER ON THE "SENSE OF EFFORT."

From further discussion with Dr. Bastian, I gather that the following are some of the chief points upon which my reasoning is, in his opinion, defective; and I have to thank the courtesy of the Editor of Brain, for giving me the opportunity of endeavouring to clear them up.

1. *Dr. Bastian objects that the "Sense of Effort" is too extensive a title, and that with the sub-title "an objective study," it indicates a confusion between subjective states and objective processes. "Sense of Effort" includes, in addition to muscular effort, efforts of recollection, attention, judgment, imagination, &c.*

I recognise the accuracy of this criticism. The subject of my paper is properly speaking "the sense of muscular effort," and does not include these more psychological and figurative uses of the expression.

I have endeavoured to avoid confusing the subjective and objective aspects of the phenomena indifferently referred to under the common term "fatigue," and have expressly and prominently set the danger signal against such an unconscious conjuring trick with words. I again submit, as one of the cardinal points of my entire paper, that to take the demonstrable signs of material changes of objective physiological fatigue as indicators to the undemonstrable material changes of subjective psychological fatigue, keeps us closer to phenomena, than the hitherto universally adopted procedure of drawing conclusions from the suggestions afforded by simple introspection and by clinical observation.

2. *Dr. Bastian's theory of kinæsthesis is not correctly represented in* §4.

I do not profess to have quoted Dr. Bastian's theory in Dr. Bastian's words, but have only given what their gist throughout his writings appears to me to be, *i.e.*, kinæsthesis in the Rolandic area, emanating from the periphery, and mainly from the muscles. But I ought, perhaps, to have found and quoted the *ipsissima verba.* Therefore :—

"We may much more reasonably and conveniently, in the face of all the disagreements concerning the 'Muscular Sense,' speak of a Sense of Movement (or in one word, Kinæsthesis (from *κινέω*, to move and *αἴσθησις*, sensation). To speak of a "Kinæsthetic Centre" will certainly be found more convenient than to speak of a "Sense of Movement Centre") as a separate endowment, of a complex kind, whereby we are made acquainted with the position and movements of our limbs, whereby we judge of "weight" and "resistance," and by means of which the Brain also derives much unconscious guidance in the performance of Movements generally, but especially in those of the automatic type. Impressions of various kinds combine for the perfection of this "sense of movement," and in part its cerebral seat or area coincides with that of the sense of Touch. There are included under it, as its several components, cutaneous impressions, impressions from muscles and other deep textures of the limbs (such as fasciæ, tendons, and articular surfaces), all of which yield conscious Impressions of various degrees of definiteness; and in addition there seems to be a highly important set of "unfelt" impressions, which guide the motor activity of the Brain by automatically bringing it into relation with the different degrees of contraction of all Muscles that may be in a state of action."[1]

Again :—

"I have proposed to include under the designation *Kinæsthesis*, or the 'sense of movement,' perhaps I ought rather to have spoken of 'sensations of movement,' as Wundt does, but the former term is more in accordance with those already in use in connection with this subject (e.g., 'muscular sense,' and 'sense of force'). Impressions of various kinds combine for the perfection of this 'sense of movement,' and in part its cerebral seat or area corresponds with the sense of touch. Thus, under it are included, as its several components, cutaneous impressions, impressions from muscles and other deep textures of the limbs (such are fasciæ, tendons, and articular surfaces) all of which yield conscious impressions of various degrees of definiteness; whilst, in addition, there seems to be a highly important set of unfelt or but little felt impressions which guide the volitional activity of the brain, in ways hereafter to be defined, and which serve to bring it into relation with the different degrees of contraction of all muscles that may be called into action."[2]

and further :—

"The most typical and important group of kinæsthetic impressions, viz., those derived from the muscles themselves, belong like visceral impressions, to the class of 'intrinsic' sensations, which, as a whole, receive but little of our conscious attention under all ordinary conditions of life. These are 'means' rather than 'ends,' and consequently soon become sub-conscious modes of activity."

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[1] "The Brain as an Organ of Mind." 1880.

[2] "Muscular Sense" debate, p. 5. BRAIN, 1887.

And in the tabulated summary of views given by Dr. Bastian at pp. 695-6 of "The Brain as an Organ of Mind," he clearly formulates his theory of the exclusively peripheral origin of kinæsthesis, in opposition to central and centro-peripheral theories. The propositions in point contained in that summary are as follows:—

"We apprehend different degrees of *resistance* and *weight* through SENSORY CENTRES. Through impressions of tension and pressure transmitted by ordinary sensory nerves from the moving members, *e.g.*, from muscles, from joints and from skin; and possibly, in addition, through certain 'unconscious' impressions passing by special afferent nerves from the spinal motor centres."

3. The third head of Dr. Bastian's criticism is more serious. *He objects, in reference to my treatment, of the clinical argument, that there is no justification for rejecting the evidence coming from patients suffering from functional hemianæsthesia, and that such rejection does not summarily dispose of evidence totally adverse to my views, and which ought to be fairly faced.*

I have already given expression to the extreme diffidence with which I have approached clinical data; in the absence of first-hand information based upon mature and cautious observation, I should hardly have felt justified in uttering any opinion on these data, if it were not that appeal has been made to them by Dr. Bastian himself as "obvious clinical facts." The evidence cannot, indeed, be fully appreciated by any but a clinical observer, capable of instinctively realising the weight of positive data in and between the lines of a clinical record. I expressly disclaim competency in that respect, and in so far do not face the evidence, but I further expressly admit that so far from disposing of the evidence this refusal amounts to accepting it as presented by Dr. Bastian. This I have, of course, no right to do; yet supposing the acceptance made, and to the full value of the terms in which the evidence is presented by Dr. Bastian, I hold myself entitled to say whether or no to my mind Dr. Bastian's premises contain Dr. Bastian's conclusion. I do not think they do, nor that the cases quoted by Dr. Bastian ("Muscular Sense," p. 11 to 20. "Brain as an Organ of Mind," p. 486) "suffice of themselves completely to displace the doctrine of Bain and of Wundt ('Muscular Sense,' p. 129), and are conclusively opposed to the notion of Wundt, Bain, and Lewes, and to others who may hold that any part of our notions concerning degrees of 'resistance' are derived from the volitional or motor centres" ("Brain as an Organ of Mind," p. 698). I do not see these cases to be logically incompatible with the conclusion I have come to from physiological data. Dr. Bastian considers it incumbent upon me to give reasons for this "non-content" as regards his (in my opinion) far too sweeping dictum that the doctrine of Bain and Wundt[1] has been "completely disproved," but in such an obscure and perplexing matter this seems to be liable to degenerate into special pleading, and not likely to promote the dissipation of obscurity. All that I think desirable or necessary with reference to the clinical argument is to indicate again the premises most relied upon by Dr. Bastian and the well-known conclusion deemed by him to be their resultant. These premises are easily accessible to the readers of BRAIN, and need not, therefore, be reprinted here.

It is hardly necessary to remind the reader that my entire paper is an endeavour to view the problem from an altogether different standpoint, and to rest the conclusion upon purely physiological data.

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[1] The names of Bain and Wundt ought not to be, as is usually the case, bracketed together in respect of the central emission theory. I have already indicated at the conclusion of my paper that this is inaccurate, and may take this opportunity of justifying the indication by the following passage from the French translation (1886) of "Wundt's Psychology," p. 421:—

"Néanmoins, les physiologistes—dans le but d'arriver à la simplicité des explications (quoiqu'ils aient échoué dans ce cas)—ont souvent essayé de faire dériver, autant que possible, toutes les sensations de mouvement d'*une seule* source. Dans cette intention, ils ont cherché 1° à les ramener aux *sensations de pression de la peau.* 2° Ils ont voulu y voir des *sensations musculaires spécifiques*, qui, dépendant des appareils sensibles et des nerfs situés à l'intérieur des muscles, seraient considérées en quelque sorte comme les sensations d'un sixième sens, du sens musculaire. 3° Enfin, selon une autre supposition, ce sont des *sensations d'innervation* : elles dépendent uniquement de l'innervation centrale des organes moteurs, et leur origine serait donc plutôt centrale, que périphérique. On voit aisément, que chacune de ces trois hypothèses "sur le sens musculaire" est insuffisante, car aucune n'est capable d'expliquer la totalité des phénomènes, que nous rencontrons dans le domaine des sensations de mouvement. En outre, chacune de ces hypothèses contient, évidemment, une *partie* de la vérité; et, par conséquent, les sensations de mouvement, comme nous l'avons montré précédemment, sont pour nous des produits fusionnés complexes, provenant de sensations d'origine différente."

The italics are in the original, and the passage is further developed in succeeding pages.

MORBID CHANGES IN THE CORTEX CEREBRI IN A CASE OF TUBERCULAR MENINGITIS AS SHOWN BY THE FRESH METHOD OF EXAMINATION.

BY EDWIN GOODALL, M.D.LOND., B.S., M.R.C.P.,

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The changes described in the pia mater and cerebral cortex by the authorities below-mentioned[1] were presumably seen in sections from brain prepared by some hardening re-agent, because in their descriptions, no mention is made—so far as I can ascertain—of certain morbid appearances which are quite obtrusive in fresh sections (æther-frozen brain, aniline blue-black). In hardened sections such appearances might well be entirely absent, or at any rate so highly distorted as to earn only partial recognition.[2] It may therefore, be desirable to refer to certain changes which fresh sections clearly show, drawing attention merely incidentally to the morbid conditions which the authors mentioned have described.

An increase of the connective-tissue elements (accountable for the abnormal degree of coloration seen) is noticeable throughout the cortex in these sections, being especially remarkable in the peripheral layer, Small round cells are numerous there, as is stated by Rindfleisch, and by Ziegler, who says "the disease of the pia mater extends to the cortex as a diffuse cellular inflammation." But a far more striking feature of the peripheral layer, and one which these writers do not describe, is the other element of the connective-tissue—the "flask-shaped" cell.[3] Whereas, in healthy sections the cells so named are barely visible, in the present ones they are seen in large numbers, and each cell is deeply stained, of large size, and possessed of numerous processes ("spider-cells"), which form a meshwork with those from adjoining cells. The morbid development of these elements explains the adhesion of the pia mater to the cortex, and the consequent facility with which a portion of membrane may be obtained with each section. Huguenin (Ziemssen's "Cyclopædia") speaks of the adhesions existing between the vessels of the pia and the cortex, owing to which strips of the latter come away on removal of the membrane; but the nature of the adhesions is not entered into. Fresh sections show that the vessels dipping into the cortex from the pia are connected on either side with long processes from the spider-cells, and that the membrane itself is still further bound down to the brain by a closely-woven tissue formed of similar processes. The spider-cells cannot be traced, in these specimens, deeper in the cortex than the third layer (that containing the large pyramidal cells), and the inference is that the morbid process had expended its chief force on the superficial parts of the brain; yet—as will appear later—in some of the specimens the large "motor" cells presented changes from the normal, so that the deeper part of the grey matter had not entirely escaped.

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[1] Rindfleisch, Rokitansky, Cornil and Ranvier, Ziegler, Huguenin, Rilliet and Barthez, Wernicke [*Lehrbuch der Gehirnkrankheiten*]; J. Seitz [*Die Menigitis Tuberculosa der Erwachsenen*].

[2] Probably excellent results are attainable by the employment of Flemming's Mixture (chrome-osmium acetic acid); but one would scarcely expect them to rival those obtained by the fresh method.

[3] Bevan Lewis: *Text-Book of Mental Diseases.*

As regards the capillaries of the cortex, they are individually prominent, and appear to be in excess. That they are so is probably the case; but doubtless many vessels barely distinguishable in the healthy cortex, by reason of their delicacy, are brought out when that part is the subject of inflammation. Some can be seen in connection with clumps of small round cells—inflammatory foci, possibly of the nature of tubercle (but similar clumps are to be seen in the cortex of the general paralytic, especially when death has supervened at an acute stage of the disorder.) Such foci of disease, together with vascular engorgement and minute extravasations, are described by all authors; here it need only be remarked, in connection with the last-mentioned, that in fresh sections, unless distilled water be used in washing, minute deposits of foreign material of yellowish-brown appearance occur, and are highly suggestive of extravasations of blood, especially when they adjoin a blood-vessel.

The state of the nerve-cells claims more attention, for hitherto it does not appear to have received much. Huguenin says he has never been able to discover changes in the "ganglion-cells." Ziegler refers to a "remarkable swelling" often noticed in them, and further, to "destruction of the nerve-elements," consequent upon cellular infiltration of the cortex. Otherwise, I have been unable to find particular mention of the condition of the nerve-cells.

In the present case, the quality of the cortex is seen to vary considerably in the different sections. They were taken from the left paracentral lobule and from the left ascending frontal convolution, near its upper end. Whereas in some sections the cortical layers, with their individual nerve-cells, are well-marked, and numerous processes can be seen streaming upward from the latter towards the periphery, in others the cortex can be roughly divided into three layers merely—a peripheral layer, of spider-cells mainly; a central, of large "motor cells," and an intermediate, in which the cells would seem to be gravely implicated. This intermediate portion includes the pyramidal nerve-elements of the second and third layers of the normal cortex, and a great number of the comparatively few cells in it are stunted, the apex-processes being either invisible or quite short; are of round or irregular shape, often showing merely a nucleus surrounded by a little cell-substance; are blurred and poorly stained, although adjacent connective-tissue cells are deeply stained; are, in fact, mere ghosts of their normal selves. Between these diseased nerve-cells are the spider-cells before-mentioned; they are often to be seen in immediate contact with a nerve-cell, partly covering it, and still further by their processes obscuring both cell and its apical prolongation. In some places an area of blurred and granular appearance surrounds the spider-cell, and has around it the diseased pyramidal cells.

Now if the views hitherto prevalent concerning the spider-cell be correct, there is nothing remarkable in the observation that, in an inflamed tissue, an enlarged connective-tissue cell and a diseased nerve cell (common results of the inflammatory process) are in juxtaposition. But if the spider cell be regarded as a "distal extension of the lymphatic system," a "scavenger,"[1] then the observation becomes noteworthy. In this case the spider cells were remarkably clear and well-developed; most of them, at any rate, showed no such signs of deterioration as are evident in old inflammatory states. Assuming, for the moment, that the views lately urged in regard to the function of these cells are correct, I apprehend that at the time of death they had but recently exceeded their normal *rôle* of depurating agents, and begun to attack the essential elements (nerve-cells), and that the day of their own degeneration was as yet distant. In connection with this supposition the duration of the disorder may be considered; death occurred precisely three weeks after the earliest symptoms were noted. The case was in all respects typical clinically, and *post-mortem* the naked-eye appearances of the brain were quite characteristic. Caseous mesenteric glands also found.

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[1] Bevan Lewis: *Op. Cit.*

As extensive changes had taken place in the region of the hemisphere which borders the longitudinal fissure, it is highly probable that, in the neighbourhood of the Sylvian fissure, the evidence of disease was still more marked. Meningo-encephalitis in the latter part would, of course, still further soften an already comparatively soft tissue; yet with due care fresh sections from this region could probably be obtained, in a case resembling the present, and should prove highly instructive.

BRAIN.

PART IV., 1891.

Original Articles.

TWO CASES OF GENERAL CUTANEOUS AND SENSORY ANÆSTHESIA, WITHOUT MARKED PSYCHICAL IMPLICATION.

BY HENRY J. BERKLEY, M.D. (BALTIMORE).

WHILE in certain forms of hysteria, melancholia, and alcoholism, passing or even continued disturbance of sensation, both of the cutaneous surfaces and special senses is by no means uncommon, profound and widespread anæsthesias without pronounced psychical alteration, belong to the most rare of all the forms of nervous disturbance.

To the few observations now existing in medical literature I desire to add two new cases that occurred in the wards of the City Asylum during my service there in the summer of 1890.

CASE I.—Mrs. R., an Englishwoman, was admitted to the Hospital Department in July, 1890, with a general disturbance of cutaneous sensation.

Family History.—The father of the patient died at an early age of consumption. At the age of seventy-four the mother was sent to an insane asylum, where she died a year afterwards (senile dementia?). Two maternal uncles, as well as two brothers and two sisters, have been confined at various times in asylums in England, but the form of mental alienation is unknown to the patient.

Mrs. R. was married early in life, and became the mother of five children, three of whom are living, and reported as being in good health. According to her own story, after she had been married for some years her husband became dissipated in his habits, and at the age of twenty-nine she contracted syphilis from him. After this event she had several abortions in succession, as well as some sore-throat and falling out of the hair.

From this time until six years ago she enjoyed fairly good health; then came an attack of acute articular rheumatism, with much joint swelling and pain. From this attack the general health never fully recovered, though there was no recurrence of the arthritis, or joint deformity. Several months later the eyesight gradually began to grow dim, and there were occasional flashes of light before the eyes; then vision was suddenly completely lost, returning in part after a few days, and was then finally and gradually extinguished. At this period she also suffered a recurrence of the sore throat, which lasted some weeks.

Her health then returned to its usual state until the Christmas of 1889, when she became troubled with a general tingling and formication of the skin of the entire body, but especially troublesome in the fore-arms and calves of the legs. These paræsthesias were soon in great measure lost, and beyond a certain inability to feel or hold objects in the hands, she was no longer disturbed.

In the first week of the year 1890, the patient had an attack of emesis, which continued through several weeks. The vomiting was incessant, and was uninfluenced by any of the ordinary remedies. Very little or no pain was present during the attack, which from the attendant physician's report seems to have been solely of a reflex nature. Periods of emesis of the same kind have been repeated at irregular intervals to the present date, gradually lessening in severity and duration.

Present State.—Mrs. R. is a slightly built, rather well developed woman of medium height. The cranium is small, but perfectly symmetrical. The hair is thin, patchy. The skin over the entire body is well nourished, yellowish in hue. The facial muscles are firm, reacting normally to the will and to faradism. The ocular muscles are perfect in their action. Those of the extremities are small, and their strength is very moderate; the hand grip is quite weak. Dynamometer R. and L., 50. There are no trophic lesions about the nails of the hands and feet.

Examination of the thorax and abdomen gave a negative result. The heart's action is normal, a little rapid—85 to the minute; tonus fair. Arteries a little thickened.

The appetite is very poor, desire for food being almost extinct. Thirst is not great. The urine contained neither albumen nor sugar, on repeated tests.

The eyesight is reduced to the perception of the difference between light and darkness in the left eye, in the right there is absolute blindness. The surface of the eye-balls and conjunctival sacs is not quite, but very nearly dry. The conjunctivæ are very slightly reddened. Both pupils are in a state of mid-dilatation, and respond very feebly to light, direct or reflected. There is no response on irritation of the skin of the neck. Headache is not complained of.

Hearing is normal, both for the voice, and for high and low pitched tones. A low ticking watch is heard at the distance of 36 cm. by both ears, also distinctly through the cranial bones.

Ammonia, iodoform, essential oils, camphor, &c., are not recognised with either nostril. Both nares and turbinated bones are dry and injected.

The buccal cavity is extremely dry, the tongue heavily furred on its dorsum, and exceedingly cracked; the epithelium is eroded from the tip and sides. The buccal mucous surfaces are red and congested. The orifices of the submaxillary ducts are raised above the surface and reddened. No secretion can be seen to escape from these ducts, and but little seems to come from the parotid. The velum palati is injected, and does not retract when irritated. There is no faucial or pharyngeal reflex, and no sensation of nausea, or act of coughing is produced when the fauces are titillated, or even when a brush is carried down over the epiglottis; yet no accident has occurred during the ingestion of food. Articulation is made somewhat thick by the dryness of the tongue and buccal cavity, but each word is enunciated clearly.

Salt and sugar solutions pencilled over the tongue, hard palate, uvula, and arches, are not perceived; over the epiglottis there seems to be some sensation. Quinine is nowhere perceived, neither are acids. With a constant current of two to three cells the result is also negative; the whole tongue, hard and soft palates, arches, back of pharynx, &c., were successively gone over without eliciting any result. There is even no sensation when the electrodes are carried behind the uvula, high into the posterior nares and pharynx.

There is absolute loss of sensation for heat, cold, pain, almost absolute for tactile and pressure sensations over the whole of the cephalic, superior extremities, trunk, and inferior extremities, with the exception of a portion of the soles of the feet. The corneæ and eye-balls may be roughly rubbed, the nose titillated with a feather, the point of a pin inserted in the tongue or finger-tips, or the skin irritated with the most violent faradic current, without producing the slightest sensation or discomfort. Over the fore-arms and hands, by repeatedly roughly pressing the muscles against the bone, or by roughly stroking the skin, the patient says that she feels "something," but cannot describe what it is. Striking a bony prominence is perceived somewhat more quickly, though the blow requires to be sharp. With the ordinary test of the anæsthesiometer, tactile sensation seems to be quite abolished everywhere in the hands; yet the patient is able, though with difficulty, to find a pledget of wool placed on a cotton cloth before her, or to pick up a glass and place it on her head, or to touch the tip of the nose with the forefinger, or unfasten a button. None of these trials succeed readily, and only after a number of attempts does success come. Bandaging the eyes seems to make but little difference. Stopping the ears with cotton wool makes the ability to pick up small objects more uncertain; it then requires larger objects, and many more contacts before the object is perceived, and renders the act of touching the nose nearly impossible. When the eyes are bandaged and the ears closely stopped, the patient becomes quiet, the respiration perceptibly lengthens, but sleep does not follow, though the experiment be continued for a number of minutes.

Ordinarily the sense of hearing is used, and very accurately, in localising the position of objects, *e.g.*, a palm-leaf fan being placed before her, she will move the hands around until it is struck somewhat sharply and a sound produced, when it is immediately grasped. Objects that produce no noise on contact are not readily discovered. A pencil placed in her hand is recognised by pressing the sharp point against a bony prominence of the finger, but any round object is mistaken for the pencil. A hat placed in her hands was not recognised for some seconds, until the hand swept around the crown, when she mentioned the word "hat" with hesitation. When the first joint of my index finger is placed on the tip of her nose, and the patient told to touch it, it is impossible for her to accomplish the feat without first striking her own forehead sharply two or three times; by doing this she seems to derive a certain sense of locality, and is then able to touch my finger once, after which she fails until the forehead is again tapped several times, then it is possible to touch the finger again.

The æsthesiometer and light stroking of the skin having failed to establish if a degree of ordinary sensibility was present or not, and it being evident from the other tests that a portion of it is retained, the following method was devised by which both observer and patient could perceive the same impression at the same moment, and the errors of the patient could be corrected to a certain degree by the observer.

The slow vibrator of a faradic battery is made to beat a minimum number of times per minute. A large electrode is taken in the left hand of the operator, who thoroughly moistens the tip of the index finger of the same hand, and applies it to the volar surface of the tip of one of the patient's fingers, where there is no muscular tissue. A small well moistened electrode is then applied to the other volar surface of the finger; then patient and observer receive co-equally the sensation of the electric discharge.

Using this arrangement, Mrs. R. says she feels a "beating something" in the tips of all the fingers of both hands at the moment of the discharge; whereas, when the electrodes are applied directly to the fingers the sensation produced cannot be described, nor can it be felt by the operator. The sensation described as a "beating something" does not appear to be a retarded one, as it is mentioned directly after the fall of the hammer.

Therefore, according to this test there remains a certain small amount of ordinary sensibility in the fingers, sufficient to enable her to pick up and hold objects, though not sufficient to accomplish many of the finer movements.

No difference is felt between ice applied to the skin anywhere (except the soles of the feet), and a temperature of 100°F. Faradisation of the skin is equally without result. The muscular sense, when the patient is reposing, is moderately well preserved. The position of the arm, when placed over the head, is most often recognised, some failures with a good many successes. The position of the feet is not so accurately known. The bed clothes are not felt, and generally when recumbent the patient appears to have an indefinite idea of her general position. When made to stand with the feet close together, she almost immediately falls backward and to the left. If supported, she sways violently, and becomes considerably agitated. The sitting position is quite comfortable.

Pressure with weights upon the forearm, the arm being on a firm surface (half to one-and-a-half kilogrammes or more) is not perceived; squeezing the muscles sharply against the bones is again described as a "something."

With weights suspended by a thread around the middle of the forearm, a difference between 80 and 112 grammes,—about one quarter—is recognised. Over the bony prominences the difference is greater, one-third not being recognised.

On the trunk, thighs, and legs, all sensations are the same as on the upper extremities. On the dorsum, sides of the foot, and all over the upper surfaces of the toes, tests are also negative. On the soles of both feet, almost symmetrically situated with each, commencing at a point about three centimetres in front of the heel and extending over the plantar surfaces of the soles and toes, sensations are almost perfect; slight touches with a needle produce a quick withdrawal; a touch upon the plantar aspect of the toes is instantly recognised and accurately located. The points of the calipers are distinguished at normal distances; heat and cold are accurately distinguished. The sensitive line passes just a little over the edges of the outer and inner metatarsal bones, where it is suddenly lost.

Locomotion is of a blind person. There is nothing approaching an ataxic gait; the woman shuffles along, seeking aid and direction from the furniture, slowly and uncertainly finding her way about. The sensibility of the soles is unconsciously used, much more than any sense of touch in the fingers.

Mrs. R. has at times a sensation of burning between the scapulæ, a girdle feeling around the abdomen, and occasionally tinglings in the calves of the legs, but there have never been any lancinating pains. There are also occasional cramps in the gastrocnemii.

Both nerves and muscles respond energetically and promptly to the faradic and galvanic currents, nor is there anything noticeable about the quality or time of the reaction. The uvula and lingual muscles respond to both currents; the uvula hangs directly in the median line. All the voluntary muscles respond to the will. When the faradic stream is passed directly through a muscle, the sensation is described as a " darting something."

The cutaneous reflexes are abolished, with the exception of the plantar, which is present and lively. The knee-jerk is present and normal, the biceps-jerk can be elicited with some little trouble. The abdominal is lost. There is one beat of the ankle joint.

The ires contract feebly with reflected light. An ophthalmoscopic examination gave the following find:—Right eye: lens very milky, so much so as to obscure the retina entirely. There is absolutely no perception of light in this eye. In the left eye the lens is somewhat milky, the fundus very slightly pink, optic nerves atrophied, vessels very few and small. The difference between light and darkness is recognised, but there is not sufficient vision to count fingers, or perceive the hand when closely approached to the face.

The patient was placed on the iodide of potassium, gradually increased to 120 grs. per diem, with but little effect, except to increase the salivary secretion, which relieved the dryness of the mouth. Mercury was also tried without result. Specific treatment after a trial of three months was abandoned, and the patient placed on bark and tonics.

During the month of September, a fibrillary clonic spasm of the facial and arm muscles began, especially marked in the adductor muscles of the thumbs; which was succeeded toward the end of the month by pronounced cramps in the flexor muscles of the arms and legs, accompanied by a considerable degree of muscular pain. There is now a good deal of sensitiveness when the muscles are pressed against the bone, which has not been noticed previously. Morphia and quinia relieve the cramps and twitchings.

The sole of the right foot has become entirely anæsthetic, the left has still a small area at the base of the great toe, where tactile and pain sensations are still perceived. This anæsthesia of the soles varies slightly from day to day, sometimes a little greater, sometimes a little less. The knee-jerk is still present.

The secretions of the buccal cavity have considerably increased in the past few weeks, and the tongue has become cleaner and less cracked.

October 18th.—The patient's general health has altered perceptibly for the worse; there is now continued diarrhœa, which does not yield to treatment. Intense abdominal and leg cramps are constantly complained of. The spreading of the anæsthesia over the soles of the feet has had the effect of confining the patient almost entirely to bed, as she is no longer able to move about without constant danger of falling.

December 9th.—Sensation has returned to the soles of the feet after having been totally absent for a number of weeks. Thermic, pain, and tactile sensibility, is now quite as acute as in July. Patient is, however, confined to the bed by the diarrhœa, which can only be relieved by a continued milk diet.

Renewed trials of the sensibility of the buccal mucous membrane shows a persistent absence of the tactile and thermic sensibility, as well as total analgesia. During the past few days transient attacks of dyspnœa, with palpitation of the heart, have occurred, especially at night. The pulse is now, during sleep, 112 per minute, regular, but rises on slight excitement to 140 beats.

July 16th, 1891.—Since the last note was taken there have been but slight changes in Mrs. R.'s condition; eyesight, smell and taste remain in exactly the same state. Hearing has altered perceptibly, high pitched notes are either not heard at all, or only imperfectly. The same watch that a year ago was distinctly heard at the distance of 36 cm. is now sometimes not perceived by either ear, even through the medium of the cranial bones; at others it is heard indistinctly a centimetre from the auditory orifices, at others only on contact. The variation is from day to day, like the anæsthesia of the soles was at first. The voice is still heard distinctly, though apparently the attention has to be constantly on the strain.

The different cutaneous and muscular sensations have remained about stationary. Tactile sensations are perhaps a trifle greater. Over the entire body, including the soles of the feet, thermic sensibility is totally absent. Analgesia to pin pricks, to the faradic current, is present everywhere, except in limited areas on the anterior portion of the soles of the feet. Tactile impressions of the rougher kinds are present in limited degree, but are evidently from the slow and uncertain movements, and the almost impossibility to touch the tip of the nose, the difficulty experienced in fastening a button, or in the more complex movements, very greatly dulled. The knowledge of the position of the limbs is even less positive than on the last examination. Differences between weights are no longer distinguished even between 60 grammes and more than half a kilo. It is no longer possible, for the patient to stand upright, nor has it been for several months.

The Strümpell experiment is still unsuccessful; with the eyes and ears closed, the woman simply becomes very quiet, the respiration deepens, but sleep does not follow.

Coarse muscular power is almost completely lost; the indicator of the dynamometer is not even moved on the scale. The response of the muscles to faradism and galvanism is prompt, and does not show any qualitative changes. Irregular muscular contractions have become a prominent symptom at times. Rapid jerkings of the head to one side or the other, or flexion of the forearm on the arm, or the leg on the thigh, occurs a few times daily.

The cutaneous reflexes are almost universally lost. The right knee reflex is uniformly not present, the left is very weak and only to be elicited with difficulty. The biceps and abdominal reflexes are absent. The right plantar reflex is also absent; the left is sometimes absent, sometimes present.

Numerous "miseries" are complained of in the limbs, though the principal pain symptom is a severe deep-seated occipital headache of a neuralgic character. Some shooting pains in the lower limbs are occasionally mentioned, but do not seem to be at all severe. The muscular spasms and pains are more intense during the night than in the daytime.

A few painless gastric crises have occurred during the winter and spring, the last being in the latter part of June; none have been severe or long continued.

The general health of the patient is not quite so good as it was a year ago. The appetite and all desire for food is entirely lost; milk is the only food that she can be induced to take constantly.

The psychical condition has undergone but slight change, she is possibly a little apathetic, with some slight tendency towards a melancholic tone, but when aroused and induced to converse for some time, this in great measure passes away. The memory is quite good.

August 15th.—A purpura simplex, in the form of bright red triangular points, has appeared over the left side of the thorax, from the fifth to the tenth rib, without apparent cause, and unaccompanied by any constitutional disturbance.

The vegetative functions, so long as the milk diet is continued, are natural. When questioned how she knows when to urinate, she replies, "I feel a pricking pain in the lower part of the abdomen." The identical pain occurs before defæcation. The urine still contains no unusual constituents; the quantity is moderate, specific gravity 1018.

The muscular sense is preserved in about the same degree as in the previous year; the position of the upper limbs is recognised, but not always accurately. Forced positions of the lower limbs are unrecognised, nor can she tell the distance the feet are from the floor when they are raised. Standing alone is quite impossible. Occasionally she will fall out of bed when attempting to move about. When supported between two persons, and told to walk, the attempt is unsuccessful, from an apparent want of knowledge of the position of the feet; they only turn and twist under her. There is sometimes vertigo, and a sensation of turning around when recumbent, nearly always in the sitting position.

A bulbous appearance is now apparent to the tips of all the fingers, but especially in the tips of the ring fingers, which has developed during the past six months. The nails are ridged longitudinally.

The heart's action is feeble; no pronounced murmur. Pulse 96; tension slight; respiration 20.

August 25th.—The purpura has entirely disappeared. Considerable deep seated pain is complained of in the mastoid processes, and post-nasal region. Pharynx very slightly congested. A sound may be carried over the epiglottis into the larynx, down the œsophagus, or upward into the posterior nares without inducing cough, discomfort, or reflex action, and without any knowledge on the part of the woman that anything is being done, other than her throat looked into.

Natural sleep is obtained in moderate amount from three to four hours during the night, seldom in the daytime.

September 16th.—The condition is unchanged since the last note was taken.

CASE II.—Mary Z., æt. thirty-five, a Russian peasant by birth, was admitted to the surgical ward of the hospital, August 2nd, 1890, for syphilis.

The patient has been in this country only about a year, and speaks very little English, making the medium of an interpreter necessary, and rendering an accurate examination in certain respects difficult. The family history, so far as can be learnt, is entirely negative. The father and mother were living when she left Russia. Patient has had good health until she contracted syphilis some thirteen or fourteen months ago. After being in the surgical ward about two weeks she became unable to walk, and my attention was directed to her by the resident physician, Dr. Hains.

Present state: August 20th.—The patient is a short, muscular, well-developed woman. The cranium is small, brachycephalic, symmetrical in contour, though the arch is a little low. The forehead is covered with a scaly purple, late secondary eruption, with some tendency to ulcerate, and much bronzing of the skin where the eruption is fading. There are characteristic specific ulcers on both tibia, also with much pigmentation. The skin from the scalp to the soles of the feet is covered with a profuse, oily, inodorous sweat, intensely disagreeable to the hand.

The abdomen shows the marks of a pregnancy. The fauces are slightly reddened, but not ulcerated. There is complaint of deep-seated pain in the thorax and abdomen. The heart sounds are normal; there are no râles or dulness on percussion. Pulse 88; temperature 99-100°F. On palpation extreme abdominal tenderness, especially localised about the pyloric end of the stomach and margin of the liver; there is also slight occipital headache. The expression is slightly apathetic when in repose; when aroused this quickly vanishes (for a few days after her confinement to the bed, patient was slightly irritable, a condition that soon passed into the usual state of cheerfulness).

The tongue is protruded straight, the facial muscles re-act normally to the will; the lips are naturally pursed in the act of whistling.

Hearing for both high and low pitched tones is normally acute. The pupils re-act naturally to light and accommodation. Vision is fair for near objects, for distant ones not so good. The fundus is apparently healthy. Colours are recognised so far as the woman is able to name them in English.

Odorous substances—ammonia, &c.—are recognised, but with extreme slowness. The mucous membrane of the nares is dry and congested. The nares may be irritated with a feather, without producing the slightest reflex movement. Gustatory sensations are absent from the anterior two-thirds of the tongue; over the posterior portion and arches present.

The grasp of the left hand is considerably stronger than the right. (The left is now always used in feeding herself, the reverse of the action when she was admitted to the ward.) The muscular tone is considerably diminished in the inferior half of the body; movements of the feet are feeble and uncertain.

The skin reflexes are either inhibited or very feeble. The plantar is absent on both sides. On the left side the knee-jerk is extremely weak, hardly to be elicited; on the right it is a little below normal. The abdominal is extinguished.

It is impossible for the patient to stand upright with the eyes unbandaged; the muscular strength is quite sufficient for her to rise to her feet, but the loss of equilibrium is immediate, and she falls backwards and to the left. Even a sitting position is to be maintained only for a few minutes; then with the arms outstretched, supporting her as props. Even then she sways violently from side to side and backwards; the face assumes an anxious expression, and if the upright position is persisted in for a few minutes longer she becomes very uncomfortable. When asked what ails her, she answers that her " head turns round."

Cutaneous sensibility, thermic, pressure, pain, tactile, is absolutely wanting with the usual tests over the whole body, except in a limited area, symmetrical on the anterior aspects of both thighs, beginning some four centimetres back from the patella, and extending over an area six centimetres broad by eight centimetres long. Within this space tactile impressions are perceived and localised, heat, cold, and pain distinguished. On every other portion of the cutaneous surface, the most intense faradic current, heat, or ice, produce no withdrawal of the extremity. Pressure on the ulnar nerves elicit none of the usual sensations of tingling in the fingers.

When the eyes are bandaged, it is impossible for the patient to discover a small object, as a coin, placed on a cloth before her; with a larger one, a water glass or india-rubber ball for example, it requires repeated sharp contacts before it is perceived, and then it is quite as difficult for her to grasp it, as it is lost as soon as found. When directed to pick up a light cotton ball, her hand having been placed on it, and put it on the top of her head, success is finally accomplished after a large number of failures. It is all but impossible for her to touch the tip of the nose with the forefinger. The attempt gives rise to a series of jerky, irregular, incoordinate movements, during which the finger may wander all over the shoulder and neck, rather than the face. Touching the tip of my forefinger placed on her nose is without success; fastening a button is impossible. So far as may be determined, the position of the limbs is totally unknown to her, and her sensation is one of floating in the air when recumbent.

The finger may be passed all over the eyeballs and corneæ without producing any reflex movements or withdrawal.

The lachrymal secretion, while scanty, is sufficient to keep the conjunctivæ in a natural condition. The oral secretion likewise is not abundant, though enough to keep the buccal cavity moist.

The facial and eye muscles respond energetically to natural and artificial stimuli. The tongue is protruded straight, without fibrillary tremor. The neck muscles respond normally to faradism and galvanism. The muscles of the left arm respond energetically to the faradic current; the muscles of the right arm very feebly. There is no difference in the reaction, or time of contraction in the flexors or extensors. With the constant stream, the left arm muscles respond naturally. The right arm muscles require a larger number of cells (three to five, Barrett) to produce a rather slow contraction, AnClC, in all the muscles. Both inferior extremities respond slowly and feebly. The muscles of the legs require forty-two cells of the same battery to produce any contraction, which is then very feeble and quite slow; AnClC in both. Fibrillary twitchings were noticed for the first time on August 31st in the inferior extremities, but only lasted a few days. The muscles of the extremities are flabby, but not atrophied.

A more thorough examination of the gustatory perceptions gives complete gustatory anæsthesia over the anterior two-thirds of the tongue. Salt is nowhere perceived when the tongue is coated with it, or when the organ is rolled around the mouth. Quinine is not perceived over the anterior two-thirds, but distinctly on the posterior third, roof of the mouth, and palatine arches. There is a slight faucial reflex and nausea when the brush is passed down upon the epiglottis. With the constant current, using two to three cells of the Barrett battery, over the anterior two-thirds there is absolutely no perception anywhere, but as soon as the region of the circumvallate papillæ is reached the patient signs that she perceives something, and when asked to tell what it is, the interpreter translates "brassy." The line between the part where sensation is present or absent, is very sharply defined; moving the double electrode a millimetre either way instantly causes a change in the facial expression, and rise of the finger. Over the roof of the mouth, palatine arches, and velum, the electric current shows that sensation is present. On the posterior wall of the pharynx there is some doubt, the patient resisting the examination. Loss of tactile sensation is apparent over the greater portion of the buccal mucous membrane and fore part of the tongue. In the posterior portions, ordinary sensibility, heat and pain sensations are present, though not very acute, though the insertion of a needle in the posterior portion of the tongue, arches, &c., produces withdrawal.

September 2nd.—The facial expression is brighter, the sensorium clearer, the eyesight evidently improved. The cutaneous anæsthesia is unchanged in general, though there is some return of sensation in the first branch of the right trigeminus. Tactile and pain sensation are perceived on the right side of the forehead and scalp, but sensation is lost exactly at the middle line.

September 9th.—Anæsthesia of all kinds is to-day complete, from the crown of the head to the soles of the feet; the sensitive areas on the thighs, and in the department of the first branch of the trigeminus, having vanished completely. The abdominal pains have to a large extent disappeared, and there seems to be no visceral complication whatever.

September 13th.—The patient occasionally sits upright by the bedside, with arms widely extended for support, but it is impossible for her to maintain the position long, the intense vertigo still continuing. The deep reflexes have altered considerably; the right knee-jerk is now a little exalted, while the left is about normal. The superficial ones cannot be elicited. The sweat secretion has lessened greatly. Smell, taste and hearing, remain as they were in August.

September 24th.—Sensory paralysis of all kinds remains the same. The intense sweating has reappeared, and become malodorous, though the external temperature is not high (68° F.). The specific eruptions and ulcers are healing rapidly under the influence of mercury biniodide and inunctions.

The reaction with the constant current is now for both arms AnClC; KClC, very weak and quite slow. Well marked AnClC and AnOC in the forearms, with slow wave reaction. In the legs AnClC, KClC, very slow and feeble. Reaction from both nerves and muscles are the same. With the faradic current there is scarcely any reply. The extremities have now decidedly less muscular force than a month before. Fewer cells are required to produce a contraction than at that time. The equilibrium is still lost when sitting upright. The knee reflex remains in the same state; there is no plantar reflex or ankle beat on either side.

Z. has had several well-marked chills, malarial in nature, on alternating days; after each the temperature would run up to 105°, and subside next morning to 101°. Administration of quinine was followed by immediate benefit.

September 30th.—The patient has improved in every way during the past three or four days. This began immediately on the subsidence of the malarial attack. She is now able to go to stool, &c., with the support of another person. The muscular tone of the legs and arms is improving. The anæsthesia remains stationary. At this date my service in the hospital ended, and I afterwards saw the case only at irregular intervals.

October 18th.—The improvement in the general health continues slowly and equally. Z. is now able to move about the room without aid, and with comparative comfort, though sudden jerks and halts to preserve the equilibrium interrupt her progress. She is yet unable to stand still without swaying violently. Tactile and pain sensations have returned in the tips of the little, index, and middle fingers of the left hand, elsewhere sensibility is unaltered. The hand grip is somewhat stronger. Both knee jerks are now present, but weak; the plantar and abdominal reflexes are absent.

During the months of November and December the return to the normal state continued gradually and progressively. Just before the return of sensation in the fingers there was the appearance of exceedingly slight thinning of the skin of the finger tips, and a transparency of the nails, but no pronounced trophic lesions. Gradually, and a little irregularly, sensation extended over the hands and feet, the muscular strength returning with it; the difficulty in preserving the equilibrium became less and less marked, and the patient was assigned some duties in the ward which she performed with diligence. Progress to health was once interrupted by a renewal of the malarial attack from which recovery was rapid, and in March, 1891, at her own request she was discharged, though there was still a considerable degree of anæsthesia over the superior portions of the limbs and trunk. Since this date the case has been lost sight of. Both knee jerks were present and normal when she was discharged. No electrical examinations were made after September. The urine always remained free from albumen and sugar. I was never able to obtain from her a correct idea of the sensations that produced the desire to urinate and defecate, the only noticeable feature about these actions being the extreme regularity at stated times in going to the commode.

The array of sensory symptoms in both cases are strikingly similar; in the first, total loss of thermic, analgesic, olfactory, gustatory, equilibrium, pressure, and weight; partial of tactile, muscular impressions, muscular sense, and auditory perceptions; uninvolved were none of the special or cutaneous sensations. Coupled with these were certain pains, mostly of a neuralgic type, located mainly in the extremities. Of motor pareses there were none.

In the second case, there was also complete thermic, pressure, equilibrium, gustatory in the anterior two-thirds of the tongue, weight, ordinary tactile and muscular feeling, almost total of the muscular sense, olfactory perceptions probably only involved through the implication of the trigeminus. Uninvolved, ocular and auditory impressions, the former perhaps a little dulled for the first few days after the beginning of the illness. Pain was also present at first, but passed away completely in a few weeks, taking rather the form of a prolonged *douleur* than a true neuralgic type. Motor pareses of the four extremities with qualitative alterations to the electric currents.

Beyond the likeness in the sensory symptoms of the two cases, there is little else in common between them.

In the first, there is a profound history of family insanity, which would suggest an instability of the nervous system of the patient. A certain amount of doubt must also be entertained of a self-given history in respect to sensory disturbances, as it is obvious that a patient is not a competent judge of many sensations. The very gradual progress of the affection during the past year, the very great masking of the sensory troubles from the woman by the eye affliction, and consequent inability to perform active movements, or attend to daily work, renders it rather a matter of suspicion whether the sensory disorders, especially those implicating the thermic and pain departments, were noticed until what I have termed gastric crises arose, which, together with the extreme dryness of the buccal cavity, caused an application to a physician for relief, and that the trouble has been one of slow and gradual progress for many months before the continued vomiting spell toward the end of 1889, possibly beginning shortly after the attack of rheumatic fever. When I first saw her in July, 1890, except for these two causes of distress, the patient was in large part ignorant of her anæsthetic condition, her only cause of complaint on that score being that she could not feel or find things so well as formerly. Partial recurrences of the paræsthesias of December, 1889, have recurred with too great frequency to allow of any weight to be thrown on that time as the beginning of the affection.

Etiologically between the first and second cases there is only one common ground of nerve disease, and that is syphilis, but with good reason for the belief it may be held that that agent had nothing to do with the causation of the anæsthesia in either observation.

In the first case, besides the direct induction of a diffuse degeneration through the agency of the syphilitic virus, we have to consider (1st) syringomyelia, which in view of the Spaeth-Shüppel observation cannot be absolutely excluded, yet the absence of muscular atrophies, and the great loss of the tactile sensibility, as well as the absence of spinal curvature, is against the theory. True, there has been some bulbous appearance lately in the tip of the finger tips, with longitudinal ridging of the nails, but these occur in other nervous affections, and in this particular instance may be referred equally well to the slight want of tone in the heart's action, with consequent defects in the circulation.

Secondly, hysteria may with certainty be excluded; the age of the patient, the equal temperament, never excited, hardly depressed; taking interest so far as her limited powers permit in whatever is going on around her, never objecting to the oft-repeated examinations, sometimes trying and exhausting to a person with so little muscular strength, does not portray a common case of the disease.

I have mentioned in the history an apathy that has been manifest in the last few months. This is only partial, and is undoubtedly connected with the diminution of the power of hearing. From it she can be easily aroused, and though her conversation is mainly about herself, she is rather contented and resigned to her fate than otherwise. What can one expect other than an apathetic condition in a person confined strictly to the bed, sans eyes, sans nose, mouth, or feeling, to carry impressions from the outer world, reduced to the sole imperfect sense of hearing? I doubt not that anyone living, no matter how naturally cheerful they may be, when placed in the same circumstances, would become more or less apathetic. Of true melancholia there certainly is none; the woman expressed her condition better than many words can tell when she once told me, "I have become a perfect dummy."

I am therefore inclined to refer the sensory state to a condition of the cerebrum approaching a psychosis—not one, so to speak, of the intellect, but an analogous condition of the sensory sphere, occurring in an hereditary *déséquilebre*, with an anatomical ground in the sensory (sensitive) cells of the cortex, fine perhaps as in the true psychoses, and as unattainable by the microscope as they are to-day, but nevertheless there, perhaps to be ascertained in future years by finer processes of hardening and staining yet unknown. In regarding the sensory disturbance as functional, I am in accord with Strumpell, Eisenlohr, Heyne, Krukenburg, and von Ziemssen.

With reference to this particular case, the least clear point in the theory is the extinction of the spinal reflexes; in Krukenburg's and von Ziemssen's cases they were either normal or increased in activity, and in Strumpell's some increased, some lost; in the present one they were all lost or greatly diminished, with the exception of some involuntary ones. With the skin reflexes this objection might be overcome, as many lesions of the cerebrum that cause hemiplegia also cause a diminution of the cutaneous reflex action on the paralysed side, but such is not the case with the muscle reflexes, and any inhibition of the cortical centres would produce overaction, not inhibition. We must therefore conjoin with the functional disturbance of the hemispheres a like disturbance of the cerebellum, which will also account, in some measure, for many other symptoms; the most prominent being the inco-ordination, muscular sense, turning sensations and vertigo.

To the present date but three autopsies have been obtained on cases of cutaneous and sensory anæsthesia—von Ziemssen's and Krukenburg's, with no pathological change in either the peripheral or central nervous system, and Shüppel's, which, as elsewhere mentioned, turned out to be a case of syringomyelia.

A coarse bilateral lesion affecting the sensory fibres, where they run closely together in the pons, peduncles, or posterior part of the internal capsules (even supposing for the moment that the paths of the olfactory, gustatory, and auditory sensations, as well as the cutaneous and muscular sensibility, run all together in this region of the brain), so limited on both sides as not to involve anything else than sensory fibres, is to my mind an impossibility, and we are obliged to turn to a functional disturbance of the cortex of the hemispheres and cerebellum, or preferably to an organic lesion of the cerebrum, to the other extremity of the nerve tracts, the terminal end apparatus.

In our second case, I think hysteria may also without doubt be excluded by the equal and cheerful disposition of the woman, as well as by the general course of the disease; rising to a certain point of intensity, stationary for a time, followed by a decadence of the acuter symptoms and a gradual recovery; but above all, by the qualitative electro-muscular changes.

An epitome of the observation gives the following: a woman in the late stages of secondary syphilis has suddenly an inability to walk from severe vertigo; there is at the same time slight fever, accompanied by headache and bodily pains of some continuance, succeeded in a few days by a notable paralytic weakening of the inferior extremities and of the right forearm, followed later by a milder implication of the left, with changes in the skin and muscle reflexes of an inhibitory character. The muscular pareses increase with the deepening of the anæsthesias, until certain qualitative changes are produced on the application of the electric currents, approximating a partial reaction of degeneration in the fifth and sixth weeks of the course of the disease ; then follows an intercurrent malarial attack, after which recovery was very gradual and progressive, sufficient to enable her to leave the hospital towards the end of the seventh month.

The main point to determine in the history is whether the muscular pareses were apart and separate from the anæsthetic affection—a motor neuritis of a mild type concomitant with another affection, or are they only separate divisions of the same disease. From their beginning together, progressing together, and diminishing together, it can hardly be considered doubtful that they were common symptoms of a single trouble, a polyneuritis of a toxic nature, occurring in the course of a syphilitic affection ; but whether caused by the specific virus of that affection, or by some other toxic material acting on an already depressed nervous system, must remain only a subject of conjecture. I cannot but consider the malarial attacks that twice interrupted the progress of the malady as anything more than a mere accidental disturbance, which seemed to have a rather beneficial influence than otherwise.

Diagnostically, but three other affections are to be thought of besides hysteria in considering the case. First, a central affection of the gray matter of the medulla and spinal cord, combined with a neuritis of the peripheral nerves ; secondly, a syphilitic exudation of a hæmorrhagic-gummatous nature, involving the spinal nerve roots from the lumbar region to the pons, and of a more intense character in the spinal than in the pontic regions; or lastly, a simple toxic neuritis having a special affinity for the sensitive fibres.

To the first hypothesis I am strongly inclined in view of the recent numerous cases of polyneuritis that have shewn, besides the peripheral lesion, an implication of the central nervous system, and especially of the posterior region of the cord.

Of mental alterations in this case there were absolutely none after the first few days of fever. The slight mental apathy and dimming of vision, followed by a slight degree of irritability, are perfectly in accord with the febrile movement; perhaps had the toxic influence been more severe, there would have occurred symptoms analogous to Korsakow's polyneuritic psychosis, and made more pregnant its relation to the psyche.

That Strümpell's sleep-inducing experiment should have been unsuccessful with R. cannot be a source of surprise. The woman had received in her youth a moderately good education in a private school in Islington, London; she is intelligent, the memory is fairly good; ordinary sensibility is to a small degree preserved; altogether the conditions differ widely from Strümpell's in a boy of fifteen years, and Krukenburg's of a very pronounced melancholic temperament, as well as from Von Ziemssen's with rapid dementia. Our patient had the memories of the past to fall back upon; the loss of eyesight in the recent years would induce her to be more reflective; besides, its constant disuse may also come in as a not unimportant factor. On one occasion I bandaged the eyes and packed the ears with cotton; the respiration deepened slightly; then I left her for ten minutes. She lay perfectly quiet, but on removing the bandages it was seen that she was not sleeping. In the second case through some lapsus the experiment was not tried.

Inco-ordination in both cases was mainly of a cerebellar type; a reeling gait, a tendency to a zig-zag direction, and oscillations in the walk, an inclination to sway violently from side to side and vertigo, were the principal features, better marked, however, with Z. than with R. Even to the latest date of the history, implication of the arms is to a large extent absent with the latter, but it was much more prominent with the other woman. In neither case was there the extreme irregularity of the movements of the legs that characterise an ordinary case of locomotor ataxy, nor the involuntary contraction and relaxation of the muscles of the hands and feet.

The preservation of the muscular sense in some part in the first case, as well as its not total abolishment in the second, is an interesting feature. With R. a well marked loss in the legs was first apparent after the commencement of the inhibition of the knee reflex, accompanied at the same time by an increase in the anæsthesia of the soles of the feet. Only at this period was the reeling gait very evident, to be succeeded before a great many weeks by a total inability to take a single step.

GENERAL LITERATURE OF CUTANEOUS AND SENSORY ANÆSTHESIA.

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ON THE LOCALISATION OF THE AUDITORY CENTRE.

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Although the localisation of the auditory centre or sphere in the first or in the two upper temporal convolutions is generally admitted, this view has not received universal acceptance; therefore, the great value of the history and autopsy here recorded will be acknowledged.

The recent views with reference to auditory localisation mostly agree in placing the auditory centre or centres in the temporal lobe, but differ as to their exact position in this lobe or as to the extent of cortex included in the auditory field. According to Munk, whose theories have been based chiefly on experiments performed on dogs, the auditory sphere is at a position corresponding in the monkey and the man to about the middle temporal gyre, considerably lower in the temporal lobe than Ferrier's localisation. In the upper or posterior portion of the superior temporal convolution, to which Ferrier gives the preference as the seat of the auditory centre, Munk localises the area for sensibility of the auricle. Luciani includes in the auditory sphere the parts which correspond to the upper temporal gyres, but, in addition, a large part of the cortex which is concerned with other functions. Schafer repeated Ferrier's experiments in a number of monkeys, obtaining some of the results of this investigator, but failing to get others. In conjunction with Dr. Sanger Brown, he, in six cases, more or less completely destroyed the superior temporal convolution on both sides, and claims that in all, hearing was not only not abolished but was not perceptibly affected. In one of the six monkeys the whole temporal lobe of the one side was removed, and yet Schafer holds that the animal responded to all impressions of all the senses, but appeared to understand very imperfectly the meaning of such expressions. Sounds even slight in intensity were heard. He believes that this case militates strongly against the view that auditory perception is localised in the temporal lobes.

Ferrier has ably replied to the attacks of Schafer both in BRAIN, April, 1888, and in the Croonian lectures.[1] He also reaffirms the accuracy of his own experiments and the justice of his own conclusions. To clear this matter up, he reinvestigated the question. In one monkey in which he performed the operation of bilateral extirpation of the superior temporal gyre, with one month interval between the operations, the result was very striking. The following, in his own words, are the conclusions as to the condition of this animal after the second operation.

"At first it failed absolutely to respond to any of the tests which invariably attracted the attention of normal monkeys. To the last, with the single exception, perhaps, of the door of the laboratory from which it was always expecting something, it never realised the origin of sounds; it was altogether indifferent to sounds which formerly were full of significance to it, and all that could be said was that it was not, when otherwise occupied, insensible to sonorous vibrations. And it would have been difficult to make this out with any degree of certainty had it not been that the animal was one qualified to give oral testimony of the fact. Whether this form of auditory sensibility is to be attributed to portions of the cortical auditory centres undestroyed by the lesions described, or to the subcortical or mesencephalic centres, I am not yet in a position to decide."

He finds further confirmation of the localisation of the auditory centre in the superior temporal convolution in cases of auditory discharges or subjective auditory sensations in connection with irritative lesions implicating this gyrus. " Gowers has reported two cases of this nature. In one a tumor, of which the oldest part was beneath the superior temporal convolution, caused convulsions commencing with an auditory aura referred to the opposite ear. In the other, a tumor affecting the superior temporal gyrus caused unilateral convulsions preceded by a loud noise as of machinery. And Hughes Bennett has reported several cases of auditory sensory discharges followed by a temporary loss of hearing in the opposite ear or in both. Thus, a woman, subject to epileptic attacks preceded by a loud noise like the ringing of a bell in the left ear, became temporarily deaf in each ear after each attack. Both ears were deficient in hearing, but the left undoubtedly more so."

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[1] The Croonian lectures on Cerebral Localisation, delivered before the Royal College of Physicians, June, 1890. From these lectures the summary of the discussion of auditory localisation is chiefly taken.

The present case should be ranked as conclusive in the decision of the question of auditory localisation, the specimens showing an insolated lesion in the two upper temporal gyres of the left hemisphere, which caused word deafness, and also a later lesion of the two superior temporals of the other hemisphere with a history of almost total deafness.

Ferrier speaks of the extreme rarity of bilateral lesions of the superior temporal convolution, but gives two important cases in which these double lesions simultaneously or successively occurred. One case, first recorded by Shaw, is that of a woman who suddenly lost power in the right arm, with also loss of speech and word deafness. The loss of power passed away, but she became incoherent and subject to delusions, and on testing her she was found to be deaf and blind. The *post-mortem* showed atrophy of both the angular and the two superior convolutions of both hemispheres. The other was a case of Wernicke and Friedlander, a woman who, after apoplexy, had right hemiplegia, aphasia, paraphasia, and word-deafness. A few months later she had a second attack, causing paresis of the left arm, and she became absolutely deaf. Extensive lesions were found in the superior temporal convolutions of both hemispheres.

I have recorded the case of a man, deaf for thirty years, whose brain showed marked atrophy of both superior temporal convolutions, particularly the left side.[1] Broadbent has recorded a similar case referred to by Ferrier.

Numerous cases of lesions of the superior temporal gyres of one side have been reported, and word deafness has most frequently been found an accompaniment of lesion of the left first temporal convolution. These facts are now well known, and have been put on record by various observers as Seppilli, Ferrier, Starr and others.

Word-deafness following an apopletic seizure—more complete deafness and partial left-sided paralysis following a second apoplexy—autopsy showing lesions of the first and second temporal convolutions of both hemispheres—a lesion of the first and second temporal convolutions of the left hemisphere, which probably accurately localises the centre for word-hearing.

The patient, a woman, aged 46 years, was admitted to the Philadelphia Hospital in August, 1891. She had a history of scarlet fever in childhood, of rheumatism many years ago, and also of old venereal disease. She had had five abortions or miscarriages, and had one living child, 23 years of age, who was deaf and weak-minded. For many years she had had valvular disease of the heart. She was right-handed. Some of the facts of her history were obtained from relatives who called to see her at the hospital, and others were procured after her death from a brother and sister-in-law, whom I sought out and carefully interviewed.

Fifteen years before her death she had an apoplectic attack which left her word-deaf but not paralysed. Prior to this first attack of apoplexy her hearing had been good, but after it she could not, by hearing, understand anything that was said to her. She could, however, hear music and sounds of various kinds ; for instance, when an organ or band had performed upon the street she at times had called attention to the fact ; and she had also come down from the second or even the third story to open the front door in answer to a knock. She could hear such sounds as a bell ringing and a clock ticking. These facts were elicited from her relatives through various statements made by them, chiefly spontaneously.

When anyone wished to communicate with her it was done by means of writing or signs, as she had fully preserved her vision and was evidently not word-blind either for writing or printing.

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[1] *University Medical Magazine*, November, 1889.

She often read the newspapers, and could do so with intelligence up to a few weeks before her death. Her sister-in-law said that several times she had heard her try to read the newspaper aloud, and in so doing she had seemed to understand fully what she read, "but made a tangle of her words." From the time of the first attack she had never been able to speak well, her words becoming "jumbled" or "tangled." From the description given of her manner of speech the defect was evidently a serious form of paraphasia and paralexia.

Her relatives spoke positively of her deafness as having been due to the "stroke;" but the apoplectic attack, although it had at once caused this word-deafness and paraphasia, had not in any way, as far as could be ascertained, affected either motion or sensation. She could write, but "sometimes mixed up her words in writing."

Nine years before her death she had another and more severe apoplexy, after which her deafness increased for sounds as well as words until it was almost total. This seizure left her also with partial left hemiplegia, chiefly affecting the arm, and in this extremity, from the description, the paralysis was more marked below the elbow. Upon examination after admission to the hospital, she was found to have some contracture at the metacarpophalangeal articulations.

Six weeks before her admission she began to have pain in the left side and abdomen, which gradually increased in severity. The day before her admission she had a chill and pain in the left shoulder and arm. For five weeks she had involuntary evacuations of urine, and for about two weeks she had been unable to retain her fæces. For these six weeks she had been much more helpless than previous to this time, often letting things drop from her hands. She seemed to suffer considerably from headache, and also slept badly.

She was first examined by me on August 24, 1891. Her condition then was one of almost complete helplessness. It was impossible to make her understand what was said to her, and so far as could be determined by repeated tests she was totally deaf; but notwithstanding her weakness, helplessness, and deafness, her face had a somewhat intelligent expression. She looked about her as if she knew what was going on. She was very emaciated; her heart's action was excited, and examination showed the presence of marked murmurs, both mitral and aortic. She became feebler day by day, and died August 28.

Autopsy.—No disease of the bone or of the dura mater was found. Even before pia-arachnoid of the left hemisphere was removed, it was noticeable that the first temporal convolution was much smaller and thinner than usual, and that at the posterior extremity of it and of the second temporal, there was a depression covering a space about seven-eighths of an inch in diameter.

The *left* first temporal convolution is remarkably small, narrow, and smooth, except at its anterior extremity. Its posterior two-thirds or three-fourths have shrunken to a thin strip. At a point about the middle of the gyre, the convolution has so disappeared as to leave only a notch and shred of tissue. Just anteriorly to this point a small annectant gyre runs to the second temporal. The attenuated appearance of this superior temporal convolution is such as to attract the attention of the most inexperienced observer of such appearances.

At a position corresponding to the posterior fourth of the second temporal convolution and the parallel fissure, the brain presented a marked depression or cavity, at the bottom of which, when the specimen was in the fresh state, was a small mass of yellow, shrivelled, puckered tissue. This was evidently the remains of an old embolic softening. The subarachnoid cavity, or cyst, which was present before the inner membranes were removed, was formed by this old necrosed area and the widened parallel fissure, this widening having chiefly resulted from the atrophy of the first temporal convolution.

In connection with the question of the part played by the second temporal convolution in cerebral audition, it is, of course, important to carefully describe the condition of this convolution. From the position of the annectant convolution, it was seen to be decidedly atrophied, and in its posterior fourth, or perhaps third, it has practically disappeared, and has been replaced by the cavity or cyst just described.

The third, fourth, and fifth temporal convolutions were undoubtedly not involved in either the softening or atrophy, as the brain was carefully studied by me in both directions, from the second temporal convolution to the hippocampal and the reverse.

Around the ascending branch of the Sylvian fissure and at the bases of the two central convolutions, much atrophy has evidently also taken place. This ascending branch of the Sylvian, instead of being a mere indentation or narrow fissure as is usually the case, is wide and gaping. The hinder portion of the third frontal, and particularly the strip of convolution between the ascending Sylvian and precentral fissure, is markedly wasted.

The retro-insular convolutions are two in number, and the posterior of the two is very small. The anterior retro-insular presents the appearance of being a continuation of the anterior half of the first temporal, and the posterior retro-insular is continuous with the posterior much-shrunken half of the first temporal.

In the *right* hemisphere was an old and very extensive hæmorrhagic cyst which completely destroyed the first, and almost completely the second, temporal gyre, the island of Reil, retro-insular gyres, the lower extremities of the central gyres, and a large extent—but exactly how much could not be determined—of the ganglia and capsules. Examination from within showed that the caudate body and the thalamus had largely preserved their integrity, and the chief interior destruction was probably of the lenticular ganglion and the external capsule. In the posterior portion of the cystic area, some gyral substance is seen, but examination shows this to be the sunken in lower extremity of the inferior parietal convolution. As, on the other side, the supra-Sylvian bordering convolutions are much atrophied. The auditory nerves were atrophied. The acoustic striæ, usually so easily seen, and often so prominent, could not be made out with the naked eye.

Slight adhesions of the left pleura were present on both sides. The lungs were œdematous and exuded a turbulent fluid on pressure. The precardial sac contained an ounce and a half of fluid. The endocardium was thickened. The mitral valve was also much thickened, the orifice being so small that it would not admit the little finger. The aortic valves were in a similar but worse condition, and a clot was found in the left auricle resembling in appearance those seen in aneurisms. The spleen was small. The kidneys were small and presented on their surface large hæmorrhagic spots. On section the surface was found to be flabby, and the cortex and pyramids much distorted. The spots on the surface had irregular shapes, having the appearance of old and recent hæmorrhagic infarcts. The liver was small and moderately firm. The ovaries were dense and sclerotic.

A study of this case justifies the following conclusions:

(1) The centre for word hearing is situated in the hinder thirds of the first and second temporal convolutions; its exact position is in a line with, or just in front of, the posterior extremity of the horizontal branch of the fissure of Sylvius. Possibly it is restricted to the second temporal.

(2) The third, fourth, and fifth temporal convolutions take no part in cerebral audition.

(3) A lesion confined to the posterior thirds of the first and second temporal convolutions of the left hemisphere will produce complete, or almost complete, word deafness, the corresponding regions of the other hemisphere remaining intact.

(4) The field or sphere for all auditory memories covers a much larger cortical area than that for word-hearing, including at least the posterior two-thirds of the first and second temporal convolutions.

(5) The auditory field and special auditory centres have their highest development in the left hemisphere, but destruction of the auditory areas of the two upper temporal convolutions of both hemispheres is necessary to complete brain deafness.

(6) A lesion limited to the centre for word-hearing and causing word-deafness will cause also paraphasia in attempts at speaking, and paralexia in attempts at reading.

(7) An isolated lesion of the centre for word-hearing, producing absolute, or nearly absolute, word-deafness, does not necessarily cause inability to recall words by other means, as, for instance, through their visual signs; in such cases probably the meaning of the word is understood, although the name cannot be properly verified in consciousness.

(8) A cerebral lesion or lesions causing word-deafness will, in time, lead to secondary atrophy of the speech and oro-lingual centres on the motor or emissive side of the brain, and also to atrophy of the association tracts between the sensory and motor hearing-speech centres.

(9) The retro-insular convolutions are anatomically and functionally closely related with subdivisions of the first temporal convolution, the most posterior of these retro-insular convolutions being continuous with the posterior two-thirds of the first temporal convolution.

STUDY OF A CASE OF BULBAR PARALYSIS, WITH NOTES ON THE ORIGIN OF CERTAIN CRANIAL NERVES.

BY HOWARD H. TOOTH, M.D., F.R.C.P.

AND

WILLIAM ALDREN TURNER, M.B., M.R.C.P.

Bibliographical.—There is much literature on the anatomical facts of this disease, mainly from the pens of German and other Continental writers. Notwithstanding the large amount of attention which has been given to the subject, much ambiguity still exists regarding the exact anatomical lesions which occur. The intention of this short biographical account is to submit a statement of the facts observed by others, so that a comparison may be readily made between these and our own observations.

1. *Distribution of the disease in the white matter of the central nervous system.*—The various statements concerning the highest limit of the disease are of profound importance. Usually the sclerosis, which involves the pyramidal tracts alone, has not been found higher than the pons Varolii. But certain observers have described conditions which are believed to signify degenerative processes in the higher parts of the cerebrum. Kahler and Pick[1] traced the disease into the crura cerebri, and observed further, an atrophy of the cerebral convolutions of the Rolandic area. Kojewnikoff[2] noted that the sclerosis stopped at the level of the pons, but he was able to trace "granular bodies," which he had also found in the sclerosed area, through the middle third of the crura cerebri, the internal capsule and the coronal radiata. He also noted

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[1] Kahler and Pick, *Prager Vierteljahrsschrift*, 1879.
[2] Kojewnikoff, *Arch. de Neurologie*, No. 18, 1883.

that the grey matter of the convolutions was of normal appearance. Charcot and Marie[1] showed, in addition to the "granular bodies," a disappearance of the pyramidal cells of the motor cortex. Lumbroso[2] saw these "granular bodies" in the internal capsule and in the cortical motor region.

In opposition to these observations, and of equal importance with them, are the negative results of competent observers, who have carefully looked for these cerebral changes and found them absent. Thus Debove and Gombault[3] searched in vain for the "granular bodies." Marie[4] failed to find any change higher than the pons Varolii. Rovigli and Melotti[5] noted the integrity of the cerebral peduncles and the internal capsule, and most recently Joffroy and Achard[6] confirm the intact condition of the pons and peduncles. In the spinal cord the sclerosis is limited to the tracts which are motor in function, chiefly the crossed pyramidal tracts, but also in some cases the direct. The degeneration is usually most pronounced in the cervical region, and there it has been found not to be sharply limited to the crossed pyramidal tract, but to extend some distance forwards into the antero-lateral area. Degeneration of the anterior ground bundle has been noted by many observers, *e.g.*, Moeli, Leyden, Flechzig, Charcot, and others.[7] Muratoff[8] found that in all cases where the anterior ground bundle was implicated the posterior longitudinal fasciculus was atrophied. This seems to favour Flechzig's view, that these two tracts are functionally analogous. Kronthal[9] noted slight degeneration of some of the fibres of the fillet, accompanied by some increase of the neuroglia. This also was observed by Muratoff.[10] A slight non-essential sclerosis of the posterior columns has been noted in some cases.[11]

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[1] Charcot and Marie, *Arch. de Neurolgie*, 1885.
[2] Lumbroso, *Lo Sperimentale*, 1888.
[3] Debove and Gombault, *Arch. de Physiologie*, 1879.
[4] Marie, *Arch. de Neurotgie*, 1887.
[5] Rovigli and Melotti, *Riv. Sperimental*, 1888.
[6] Joffroy and Achard, *Arch. de Med. Experimental*, No. 3, 1890.
[7] Quoted by Muratoff, *Vide infra.*
[8] Muratoff, *Neurol. Centralblatt.*, 1891, p. 513.
[9] Kronthal, *Neurol. Centralblatt.*, 1891, p. 133.
[10] *Loc. supra cit.*
[11] Mentioned by Dr. Ormerod in a critical digest of Amyotrophic Lateral Sclerosis, BRAIN, vol. ix., p. 245, to which we are indebted for much valuable information.

2. *Distribution of the disease in the central grey matter.*—This consists of atrophy and disappearance of the multipolar nerve cells, which are included in Dr. Hughlings Jackson's "lowest level" (the bulbo-spinal centres), with the important exception that the oculo-motor nuclei are usually not degenerated. The greatest amount of atrophy is in the hypoglossal nucleus. The cells of the corpus dentatum of the inferior olive have been found affected by only one observer.[1] The nucleus ambiguus was found atrophied by Kronthal;[2] usually it is unaffected, as also is Clarke's column.

3. *Condition of the peripheral nervous system.*—Degenerative changes have been observed in the peripheral nerves in some cases. Joffroy and Achard[3] noted a parenchymatous degeneration of the nerves of the extremities in a case of this nature. Kronthal (*loc. cit.*) describes degeneration of several cranial nerves, while the peripheral spinal nerves were unaffected. Oppenheim,[4] in a case of chronic anterior poliomyelitis, found slight degenerative change in the mixed nerves, while the anterior roots were healthy. But it must be borne in mind that in many instances no change has been found in the peripheral nervous system.

4. *Nature of the change in the central nervous system.*—The sclerosis is in appearance similar to that of descending degeneration resulting from a cerebral lesion. That it is not due to a diffuse chronic myelitis is evidenced by the fact that the periphery of the cord, which is usually affected by the inflammatory condition, here completely escapes, and also the posterior columns. Whether the change be originally parenchymatous or interstitial is difficult to determine, as in the later stages the resulting sclerosis is the same; and early stages of this disease do not, as a rule, admit of pathological examination. Leyden observed that the axis cylinders were never hypertrophied, as occurs in disseminated sclerosis.

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[1] Maier: *Virch. Archiv.*, vol. 61. (Quoted by Ormerod, *loc. cit.*)

[2] *Loc. supra cit.*

[3] *Loc. supra cit.*

[4] Oppenheim: *Arch. fur Psych.* Bd. xix.

Much uncertainty exists regarding the starting point of this disease. In "pseudo-bulbar paralysis" double lesions of a hæmorrhagic nature have been found implicating the pyramidal tracts above the pons, and giving rise to secondary degenerations on both sides. In the chronic progressive form under discussion, we have been unable to find any primary lesion recorded which would account for the more or less symmetrical nature of the sclerosis. Although some observers have noted changes of a degenerative nature in the large cells of the motor cortex, many cases have been recorded where no such alterations have been detected. The view that the disease is a primary affection of cells and fibres, motor in function, seems the only explanation which our present knowledge can justify.

We are indebted to Dr. Hughlings Jackson for permission to record the following case:—

CLINICAL SUMMARY.—*Onset with "loss of voice," shortly succeeded by hemiplegic weakness of right side and later on difficulty in swallowing. Admitted with paralysis of tongue, lips, palate, and vocal cords, weakness and atrophy of right arm and leg, and inability to articulate. Later on paralysis of right side and paresis of left, paralysis of the neck muscles and muscles of deglutition. Finally, paralysis of diaphragm. No affection of eye muscles and sphincters. Duration, about sixteen months.*

G. W. V—n, ætat. 43, was admitted into the National Hospital for the Paralysed and Epileptic, Queen Square, on October 8th, 1888, and owing to his inability to articulate, and difficulty in writing, a very brief history was obtained. In December, 1887, he began to "lose his voice," but shortly after this he noted a gradually increasing stiffness and weakness of the right arm and leg; he does not think that the left side of the body was affected at this time. Some five months later, in April, 1888, he complained of considerable difficulty in swallowing and inability to expectorate effectually. He has suffered no pain during his illness. He denies syphilis. He said he had rheumatic fever nine years ago.

Condition on admission.—The body is emaciated, the expression is characteristic of the disease. This has been well described as "sad inanimate" while the temperament is buoyant and expectant. The movements of the face are weak on both sides, the orbicularis oris being especially affected, so that he is quite unable to whistle or put his mouth into the position for kissing. The upper facial group, frontalis, and corrugator supercilii is in a state of over-action, so that the forehead is crossed by deep wrinkles; the orbiculares palperbrarum act well and efficiently; the naso-labial fold is prominent on both sides. The lower facial group is weak; the lower lip hangs down and the mouth is slightly open. The tongue lies flaccid on the floor of the mouth and cannot be protruded beyond the dental arch: he is quite unable to articulate, an endeavour ends merely in the production of a noise. He understands everything that is said to him, he answers questions in writing, and he reads and appreciates the daily papers. Mentally he is facile, laughing and sobbing on slight provocation. The vision is good, the fundus of the eye is normal and there is no defect in the movements of the eyes. Hearing also is good. Dr. Semon examined the larynx and reported that there was bilateral and symmetrical paresis of the abductors; the glottis on inspiration did not open to more than three mm., on phonation there remained a small oblong gap between the cords (paresis of the internal tensors). There is paresis of the soft palate. He swallows pulps more readily than fluids or solids, but the taking of food usually starts a troublesome cough: this difficulty in swallowing appears to be due to an inability to get food into the lower pharynx. The vomiting and swallowing reflexes are not obtained on tickling the back of the tongue. The cardiac and respiratory functions are normal.

He walks with a feeble, hemiplegic gait. There is considerable wasting of the muscles of the right arm and hand, especially of the interossei and thenar groups. All movements are effected but feebly, except that he is unable to dorsiflex the right foot. In the right leg there is no marked wasting. The tendo Achillis is rigid. Both knee jerks are exaggerated, the right being more marked than the left.

An ankle clonus is not obtained: the plantar reflexes are active, the left more than the right. Defæcation and micturition are normal. The urine contains neither albumen nor sugar. The arteries are not atheromatous. *Electrical reactions.*—All the muscles react to the faradic current; but there is a quantitatively diminished reaction of the muscles of the right side in comparison with those of the left.

Progress.—Only a summary of the notes taken during the remainder of the patient's life is given here. By December the atrophy and paralysis had considerably advanced, the small muscles of the left hand being more atrophied than on admission. The faradic excitability of the right hand muscles had also correspondingly diminished; he could barely hold a pen in his right hand. In January, 1889, it was noted that the whole of the left side was becoming weaker and that he had great difficulty in chewing owing to paresis of the jaw muscles. At the same time a slight ankle clonus was observed on the left side, there was none as yet on the right. The condition of the hands was as follows:—Atrophy of the dorsal interossei and abductor indicis as well as of the thenar and hypothenar muscles, the position of the fingers is that seen in ulnar paralysis: the muscles all presented increased mechanical irritability. No fibrillary tremors were observed. At Dr. Jackson's suggestion the urine was repeatedly tested for the presence of sugar, but at no time, not even to within a few days of death was any observed. At no time during the course of the disease did the saliva trickle from the angles of the mouth.

In February the movements of the neck muscles were found to be feeble, flexion of the head backwards and forwards was effected for a short distance by muscular action, beyond this the head fell backwards or forwards by its own weight; rotation was, however, well carried out. Movement of the head occasioned considerable pain over the position of the Atlas. During the remainder of this month and the following the patient was unable to move in bed without assistance. Towards the end of March, the following condition was noted:—

Olfactory Nerve.—Sense of smell perfectly normal.

Optic Nerve.– Sight unimpaired; discs normal.

Oculo-Motor Nerves.—All ocular movements well carried out, but some coarse nystagmoid jerkings were occasionally observed on extreme lateral deviation; pupils medium and equal, react to light and accommodation; no ptosis.

Trigeminus (Motor branch).—Paresis, almost amounting to paralysis of masticatory muscles.

(Sensory branch.)—No impairment of sensation. Taste normal.

Portio dura.—Lower facial muscles completely paralysed, the upper group is in a tonic state of overaction.

Portio mollis.—Hearing is unimpaired.

Glossopharyngeus.—Vomiting reflex is in abeyance.

Vagus and accessorius vagi.—Paralysis of the abductors of the vocal cords. Paralysis of articulation. Paresis of the soft palate.

Hypoglossus.—Glossoplegia with wasting.

Spinal accessory.—Paresis, with wasting of sterno-mastoid muscles and upper parts of both trapezii.

Cervical nerves.—Paralysis of the muscles which move the head; paralysis of the diaphragm; atrophy of the infra-hyoid muscles.

Brachial plexus.—Paralysis and atrophy of the muscles of the right shoulder, arm, and hand; rigidity and increased tendon jerks; paresis and atrophy of the left arm and hand; paralysis of the right serratus magnus, paresis of the left; atrophy and paresis of both pectorales. No affection of sensation.

Dorsal nerves.—Paresis of the abdominal and back muscles.

Lumbar plexus.—Paralysis, atrophy, and rigidity of the muscles of the right leg; paresis of the left leg.

Sphincters.—Unaffected.

April 1st, 1889. *Exitus letalis.*

Autopsy.—Dura mater slightly adherent to calvarium; considerable opacity of the arachnoid over the hemispheres, but not over the base; no excess of cerebro-spinal fluid; no *naked eye* changes in brain or spinal cord.

Lungs, liver, and kidneys were of normal appearance.

Heart: slight, easily torn adhesions between epi- and pericardium.

The muscles were all of good colour, slightly soft, but not friable; they were all more or less atrophied. There was complete absence of subcutaneous fat. The platysma myoides were pale, but well developed on both sides.

MICROSCOPICAL EXAMINATION OF BRAIN, MEDULLA OBLONGATA, AND SPINAL CORD.

Vertical transverse sections of the brain were made at levels, which included part of the motor region, and carefully examined with a view to finding some alteration of the cortical cells, but no trustworthy results were obtained. Allowing for the influence of *post-mortem* decomposition, the appearance did not seem to differ much from the normal, but it was remarked that the cortex generally was very vascular.

The internal capsule is, unfortunately, affected by *post-mortem* softening in places, but those regions of it which have escaped seem to be natural.

The crura cerebri at the level of the third nucleus show signs of decomposition, which make it difficult to say whether, or not, there is any degeneration of fibres in the motor tract.

The third nucleus is not deficient in ganglion cells, though the cells themselves seem to be highly pigmented. The posterior longitudinal bundles are normal. Some sections were made of the upper pons, which showed marked degeneration of the pyramidal tracts, but not so complete as in the next section.

Unfortunately the region intervening between the last section and that at the level of the fifth nerve is missing, so that our next section is taken through the pons at the lowest level of the fifth nucleus. Here sections were made, which include the middle peduncle of the cerebellum, and, in fact, all the white matter of the cerebellum, and also the nucleus dentatus. The white fibres of the central parts of the section show the beaded appearance of *post-mortem* decomposition, but the peripheral parts are in good histological preservation. Only the lowest part of the motor nucleus of the fifth nerve can be examined in these sections, and in it can be seen only two or three ganglion cells, but several carmine-stained patches indicating degenerated cells. What can be seen of the sensory nucleus is natural. The other nuclei to be seen in this section are the superior olive, the nucleus trapezoides, and the nucleus dentatus of the cerebellum, all of which appear to be natural. The condition of the pyramidal bundles is very striking. To the naked eye, after Weigert-Pal treatment, the pyramidal regions appear very lightly stained, while under the microscope there can be seen no normal large or medium medullated fibres, and much fewer than normal small fibres. It is possible that decomposition may be accountable for a certain amount of the myeline *débris* lying about, but the transversely crossing fibres, which are numerous, show little or no decomposition. Under a high power double stained sections of the pyramidal bundles show a loose granular ground substance, plentifully besprinkled with nuclei, a few rather large droplets of blue-staining myeline matter, and a number of small black dots indicating small medullated nerve fibres or their remains. In some parts the granular ground substance is more condensed, forming a true sclerosis, but this is more noticeable in the cord than in the medulla. We may say, therefore, that the whole of the pyramidal tract is completely degenerated on both sides. There appears to be no affection of the transverse fibres forming the trapezoid body so-called. The fillet appears to be unaffected by disease, but its fibres are much beaded by decomposition, owing to delayed penetration of the hardening reagent. The posterior longitudinal fibres present quite normal appearances.

The next section includes the nuclei of the sixth and seventh nerves, and the intra-medullary course of these nerves, and the eighth. The sixth nucleus, Fig. 1, VI. n., is perfectly natural in appearance. The fibres of the sixth nerve are also natural.

The seventh nucleus, VII. n., shows very few ganglion cells, and these are abnormally rounded, atrophied or granular. The individual fibres of the seventh root in its course through the medulla seem to be quite natural, that is, none are to be seen in the process of degeneration, but the bundle, as a whole, is decidedly smaller than natural. On the other hand, the compact little bundle of transversely-cut medullated fibres, the ascending loop of the seventh Fig. I., VII. a., which is usually so conspicuous above the posterior longitudinal bundles, is here invisible by Weigert-Pal staining. On carefully examining it with higher power we find that normal nerve fibres are completely absent from it, and that it consists of loose meshwork of neuroglia and nuclei. This taken with the much less atrophied condition of the issuing seventh root is remarkable, and will be considered further on. The degeneration of the pyramidal tracts is as in the preceding section.

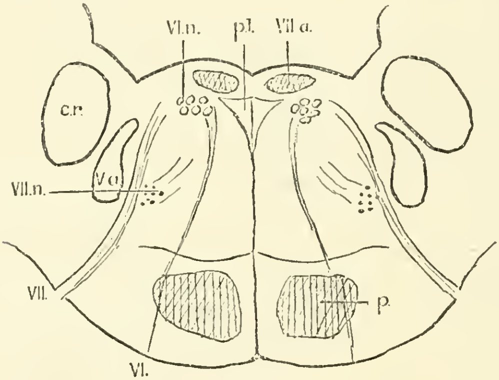

FIG. 1.

Diagram of lower pons, showing sixth and seventh nerves and nuclei, p. l., posterior longitudinal bundles, p. pyramidal tract, VII. n., seventh nucleus degenerated, VII. a., ascending loop of seventh root, VI. n., nucleus of sixth nerve, c.r., restiform body, V.a., ascending root of fifth nerve. In this and the succeeding diagrams, normal white and grey matter is drawn in outline without shading. Degenerated tracts are cross-hatched. Nerve nuclei containing healthy ganglion cells are represented by groups of small circles, those containing degenerated cells by groups of black dots.

At the junction of the pyramids with the pons the seventh nucleus is still visible, and at this level, though much affected, presents more nearly normal appearances than it did higher up, that is, there are a few normal ganglion cells. The pyramidal tracts are totally degenerated, contrasting with the healthy arciform fibres surrounding them. The eighth nucleus and nerve are natural. The restiform body and the ascending root of the fifth and eighth nerves are here, as elsewhere, normal.

The next cranial nerve to be examined is the ninth. The nucleus seems to be perfectly natural, and the same may be said of the ascending root. At this level also the nucleus ambiguus, a group of cells lying on the inner side of the ascending root of the fifth, presents quite natural appearances, except for the pigmentation of its cells, which is so common an appearance that it can scarcely be considered pathological.

The freedom from disease of the nucleus ambiguus is important because this nucleus is evidently partly, if not entirely motor, by the character of its cells, and it is believed to be the motor nucleus of the vago-glossopharyngeal group. The nucleus centralis of Roller is natural.

In every case of bulbar paralysis the condition of the hypoglossal nucleus is of great interest, and here demands detailed examination.

When making sections backwards from the brain to the spinal cord, the normal hypoglossal nucleus begins to appear at just about the level of the most inferior striæ medullares. It is at first only indicated by a few large multipolar cells scattered among the small round cells which belong, probably, to the eighth and ninth nuclei on the one side, and to the nucleus funiculi teretis on the other. As we proceed spinal-wards, however, the nucleus becomes more and more defined, lying close under the floor of the fourth ventricle, and consisting exclusively of large multipolar ganglion cells. Ventrally to the nucleus pass out the fibres of the hypoglossal nerve and close under the nucleus among the root-fibres lies another diffuse collection of small cells, the small-celled nucleus of the hypoglossal (Roller). In well-stained sections there is a close network of medullated fibres all through the chief nucleus. In each section of the nucleus when at its fullest size may be counted from thirty to forty ganglion cells, diminishing to about twenty at the level of the calamus scriptorius, and still further back to five or ten, that is, at the level of the lower or spinal extremity of the olive, just below which the nucleus disappears. The small-celled nucleus is most distinct in the cerebral or upper third of the chief nucleus, and becomes less marked as we go backwards till it disappears. It has several large cells scattered about in it.

Fig. 2 shows the relations of the nucleus in the medulla at a level at which it is usually best seen, that is, about its upper third. At the most cerebral end the nucleus contains a considerable number of normal cells. A little further back, however, this is not the case. Here, where there should be counted from thirty to forty well-formed, large multipolar ganglion cells, there are to be found in some sections perhaps as many as ten at the most, generally four or five, and in some even none at all.

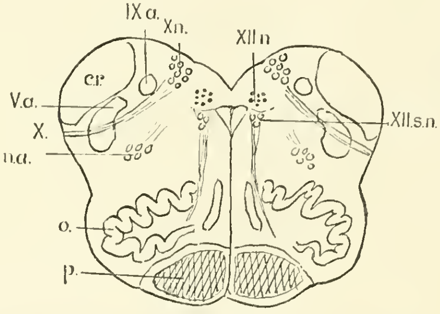

FIG. 2.

Medulla at middle olive, or lower part of floor of fourth ventricle, showing relative position of vagus and hypoglossal nuclei. IX.a. so-called ascending root of glossopharyngeal. X.n. vagus nucleus. XII.n. hypoglossal nucleus degenerated. O. olive. XII.s.n. so-called small-celled nucleus of hypoglossal (Roller) n.a. nucleus ambiguus. Other lettering as in Fig. 1.

The ground substance of the normal nucleus should be fairly compact, finely granular neuroglia supporting numberless interlacing white fibres which come out very distinctly by Weigert-Pal staining. In the affected nucleus, however, the supporting tissue consists of a loose meshwork of fine fibrils with many nuclei. There are some white medullated fibres but not so many as in the normal nucleus. The root bundles of the nerve contain decidedly fewer fibres than natural, in fact the hypoglossal roots are not the conspicuous objects that they are in sections of natural medulla oblongata.

The small-celled nucleus does not appear to be affected in any way, and among its cells may be found many normal large cells.

The vagus nucleus (X.n.) is normal.

Below the calamus scriptorius, where the central canal is completely surrounded by the central grey tube, the nucleus lying dorsally to the hypoglossal is that of the eleventh nerve or accessorius vagi. This nucleus is quite normal in appearance. The nucleus lateralis presents no sign of degeneration.

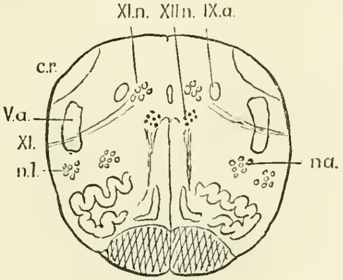

FIG. 3.

Medulla at lower olive below calamus scriptorius showing the relative position of accessorius and hypoglossal nuclei. XI.n. accessorius nucleus. n.l. Nucleus lateralis.

In the lowest part of the medulla (Fig. 4) just above the decussation, we find a great degeneration of the anterior horns (c.a.) which here first make their appearance. The hypoglossal nucleus is still visible and degenerated. The nucleus funiculi gracilis, and nucleus cuneatus internus (n.c.) and externus (seen in sections higher up than this) are quite natural. The pyramids at this level show considerable numbers of natural nerve fibres both large and small.

In the spinal cord at the first cervical nerve the grey matter of the anterior horn is almost devoid of ganglion cells, in some of the sections none are to be seen at all, in others at most two or three. The lowest fibres of the anterior pyramids are still to be seen crossing to form the crossed pyramidal tract which is now seen to be distinctly affected. It contains very few large nerve fibres compared with the healthy cord, so that by Weigert-Pal staining it appears as a lightly-stained patch, but by carmine staining it comes out a dark patch owing to a slight condensation of the neuroglia.

At cervical ii., besides disappearance of ganglion cells, there is also marked wasting of the horns, rather more marked on the left side. The lateral horns share in this wasting. There are a few natural bipolar cells in the base of the posterior horn, the position taken lower down by the posterior vesicular column. The degeneration of the crossed pyramidal tract reaches to the periphery of the section in the region of the posterior horns. There is well marked affection of the anterior median column and the direct pyramidal tract.

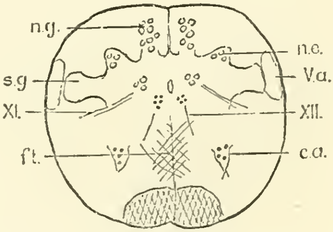

FIG. 4.

Medulla at level of first appearance of anterior horns of cord. c.a. Anterior horns, ganglion cells degenerated or absent. s.g. Substantia gelatinosa. n.c. Nucleus cuneatus internus. n.g. Nucleus funiculi gracilis. f.t. Decussation of fillet, natural.

At the level of cervical iv. (Fig. 5), the shrinking of the anterior horn brings into prominence the lateral horn, particularly on the left side. Here are a few cells, but there are the remains of many degenerated cells lying about.

There is still a very small strip of degeneration on each side of the anterior fissure (d.p.t.).

At cervical vii. the attenuation of the anterior horns is very marked, especially on the left side. The left lateral horn stands out in marked prominence, and is seen to contain many natural ganglion cells.

At dorsal ii. we have the first appearance in this series of the posterior vesicular column. This appears to be perfectly natural here and in all the sections below this level. The wasting of the anterior horns is slightly more pronounced on the left side. The crossed pyramidal tract is extensively affected, but it shows a fair sprinkling of normal large white nerve fibres scattered through it.

At dorsal vi. the wasting of the anterior horns is extreme. The degeneration of the crossed pyramidal tract now reaches the edge of this section. It has here the appearance of a sclerosis, but has in it many quite normal large nerve fibres, many more than in the last section. These fibres are, many of them, probably ascending from the posterior vesicular column to gain the direct cerebellar tract.

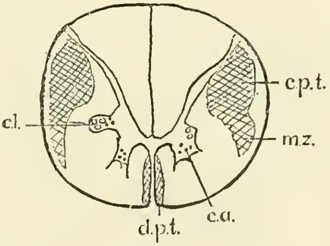

FIG. 5.

Transverse section of spinal cord at level of cervical iv.; c.p.t., crossed pyramidal tract degenerated; d.p.t., direct pyramidal tract degenerated; m.z., descending fibres in the "mixed zone" degenerated; c.l., lateral horn with some normal cells, but many degenerated.

At dorsal viii. (Fig. 6), the thinning of the anterior horns is, perhaps, at its maximum. In some of the sections a ganglion cell or two may be seen, but in others none at all. The posterior vesicular column is unaffected, contrasting strongly with the wasted anterior horns.

At lumbar i., though the anterior horns are smaller than usual, they contain a considerable number of healthy ganglion cells. The sclerosis of the crossed pyramidal tract is most pronounced on the left side. The posterior vesicular column is fully developed and perfectly normal.

At lumbar iii. (Fig. 7), the grey matter presents more nearly normal appearances than in any of the preceding sections, but even here the ganglion cells are below the normal in point of number.

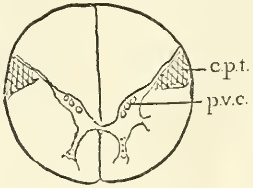

Fig. 6.

Transverse section of cord at level of dorsal viii.; p.v.c., posterior vesicular column.

The anterior root fibres proceeding from the anterior horns in all these sections appeared to be rather thin and to retain the Weigert-Pal staining badly. We hesitate, however, to pronounce this as a sign of atrophy of the fibres, because it is an appearance common in lightly stained sections of normal spinal cord. The transverse sections of the anterior roots themselves, which could be seen in most of the sections, were critically examined with absolutely negative results. It is difficult to believe that any considerable disappearance of white fibres could fail to be discovered in such a large number of sections.

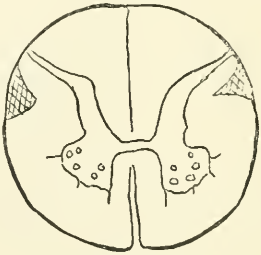

Fig. 7.

Transverse section of cord at level of lumbar iii.

Summary of Microscopical Appearances.

1. Motor cortex apparently normal.

2. White fibres of internal capsules and crura cerebri also normal.

3. Pyramidal tracts of upper and middle pons deeply degenerated, showing complete absence of white medullated fibres, and some condensation of neuroglia base.

4. Crossed pyramidal tracts both degenerated all the way down the cord, but from the decussation downwards there are some healthy white fibres scattered about in the sclerosed area.

The direct pyramidal tract is degenerated as far down as cervical iv.

5. Cranial nerve nuclei affected are:—

(*a*) Motor nucleus of fifth.

(*b*) Nucleus of seventh extensively, issuing roots attenuated only, but ascending loop deeply degenerated, being quite devoid of medullated nerve fibres.

(*c*) Hypoglossal nucleus extensively, roots much thinned.

6. Cranial and other nerve nuclei not affected by disease are those of the third (fourth not examined), sixth, seventh, ninth, tenth, eleventh, and small-celled nucleus of twelfth. Also nucleus dentatus, nucleus trapezoides, nucleus centralis, nucleus ambiguus, nucleus lateralis, nucleus arcuatus, nucleus funiculi teretis, olivary body, superior olive, nucleus funiculi gracilis, and nucleus cuneatus internus, and externus.

7. Grey matter of cord from cervical i. to lumbar i. shows extreme atrophy, with almost complete loss of ganglion cells, in the anterior horns especially, and to a very great extent in the lateral horns also. The issuing anterior root fibres appear rather thin, but the anterior roots outside the cord are normal.

8. The posterior vesicular column is everywhere normal.

The following points appear to us to be worthy of special remark:—

Distribution of disease in motor grey matter.—All the ganglionic cell groups innervating the somatic muscles, that is those nervous centres included in Dr. Hughlings Jackson's "lowest level," are deeply degenerated, with the exception of the oculo-motor nuclei, and those which control the sphincters ("escape of eyes and perinæum").

Distribution of disease in motor white matter.—There is intense degeneration of the pyramidal tracts in the middle pons, and to a lesser degree of the upper, but lower down, that is at the level of the olive, these tracts show considerable numbers of what may be healthy fibres, and also numbers of small medullated fibres. Below the decussation, the area of sclerosis differs in no respect from an ordinary descending secondary degeneration. The extension forwards of the degeneration into the region of the "mixed zone" is not due to diffuse myelitis, but as has been shown by one of us,[1] marks the normal position of certain of the descending motor fibres.

These facts suggest the possibility of the existence of a primary lesion of the pyramidal tracts in the pons with corresponding secondary descending lesions.

Condition of the peripheral nervous system.—As pointed out in the pathological report, though the ganglion cells of the anterior horns of the cord and medulla are deeply degenerated, yet the anterior nerve roots are perfectly healthy. On the other hand, the roots of the hypoglossal and facial nerves are very much thinned by the disappearance of many nerve fibres, particularly in the case of the former, but those which are present are natural in appearance. We can offer no explanation of this apparent anomaly.

Relation of facial nerve to seventh nucleus.—We are enabled by the microscopical examination of this case to produce anatomical evidence of the possible innervation of the orbicularis palpebrarum by the oculo-motor nuclei. As above described, the oculo-motor nuclei (third and sixth, fourth not examined) are quite healthy, while the seventh nucleus, and the ascending loop of the nerve (Fig. 1, vii. a.)

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[1] *Tooth.*—Secondary degeneration of the spinal cord, 1889, p. 36. Figured also by Hadden and Sherrington in BRAIN, Vol. VIII. p. 502.

lying dorsally to the posterior longitudinal bundles are deeply degenerated. Now it is probable that this ascending limb includes all the fibres from the nucleus proper. But the nerve root, on the other hand, passing through the medulla to issue from the side as the seventh nerve, though not presenting the size and compactness of the normal root, yet contains many perfectly normal fibres. We must suppose that these have come from some other source than the highly degenerated nucleus, and we believe that they come from the oculo-motor nuclei by way of the posterior longitudinal bundles, and are passing in the trunk of the facial nerve to supply the unaffected muscles of the upper face, or, as we may call them, the oculo-facial group (frontalis, corrugator supercilii and orbicularis palpebrarum).

In support of this view we refer to a suggestive paper by Mendel,[1] who performed experiments after Gudden's method on rabbits and guinea pigs. The upper and lower eyelids were stitched together, and the frontalis extirpated, in new-born animals. After six to ten months they were killed. Careful examination of the abducens and seventh nuclei failed to show any change in them. The facial trunk on the side operated upon appeared thinner than on the other side. Marked changes were found in the hinder parts of the third nucleus, namely, great diminution in the number of the ganglion cells, so as to cause the nucleus, as a whole, to be much smaller on the same than on the opposite side. The cells were atrophied and their protoplasm stained less vigorously than normal. He suggests that the path of connection between the oculo-motor nuclei and the facial nerve is the posterior longitudinal bundle.

There is reason to suppose that another important set of fibres, which reach their destination by way of the facial nerve, namely, those which supply the orbicularis oris, have their nucleus of origin in other than the seventh nucleus, probably the hypoglossal. The clinical facts of all cases of bulbar paralysis lend support to this view; they all show the very close relations which exist between the lips and the tongue. But in order to demonstrate the separation between the motor innervation of the lips from the other facial muscles, we refer to a case, mentioned by Dr. Gowers,[1] of poliomyelitis affecting the seventh nucleus only, where the orbicularis oris escaped. Our case does not add any anatomical evidence in solution of the problem as to how the fibres which supply the lips reach the facial nerve trunk from their nucleus of origin. The path is possibly the lower part of the posterior longitudinal bundles, but our case throws no light on this point.

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[1] *Mendel.—Ueber den Kernursprung des Augen-Facialis; Neurolog. Centralbl.*, 1887, p. 537. Mendel refers to a discussion on the subject in the *Berlin. Klin. Wochenschr.*, 1887, No. 48.

Relation of eleventh nerve (accessorius vagi) to hypoglossal nucleus.—This nerve is usually described as consisting of two parts, that accessory to the vagus, accessorius vagi, or eleventh nerve, and a spinal division, or accessorius spinalis. About the latter we have nothing to say, its origin, distribution, and function being fairly clear. But as to the former we find very little information, even in the latest text books. According to Lockhart Clarke, and Schwalbe, the accessorius vagi nucleus is the lowest of the three nuclei contained in one cylinder of grey matter, which nuclei are from above downwards the glossopharyngeal or ninth, the vagus or tenth, and the accessorius vagi or eleventh. As it is impossible to differentiate the lower fibres of the vagus nerve roots from those of the accessorius vagi, as they issue from the medulla between the restiform body and the olive, except inferentially by their position, so no hard and fast line can be drawn between the nuclei of origin of these nerves. But for the purpose of description it may be convenient to state that so long as the central canal is surrounded by grey matter, the ganglionic cell group lying *dorsally* to the hypoglossal nucleus, may be regarded as that of the accessorius vagi. Where, however, the canal has opened out into the fourth ventricle, the cell group lying *externally* to the hypoglossal nucleus, and thus forming part of the floor of the ventricle, is that of the vagus. This, so far as we can make out from authors, is the present position of anatomical knowledge on the subject.

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[1] Gowers' "Diseases of the Nervous System," 1888, vol. ii., p. 222.

In the case of V——n, we can say that the common nucleus of the ninth, tenth, and eleventh nerves is quite healthy. (This appears to be the condition in all recorded cases in which the state of the nucleus is specially noted, with one exception.[1]) This is at variance with the fact that he had paresis of the soft palate, and paralysis of the abductors, and adduction of the vocal cords, which muscles are regarded as being supplied through the vagus by its accessory branch. That the eleventh nerve is the path by which motor fibres reach the palate and vocal cords was long ago demonstrated clinically by Dr. Hughlings Jackson, and has been recently corroborated by experiments of stimulation and section of that nerve within the skull.[2] As we have just said, the eleventh nucleus was in our case healthy, we must therefore look elsewhere for the nucleus of origin of its motor fibres. The nucleus ambiguus from its position and connection would, at first sight, appear likely to be the motor nucleus in question, and indeed has been called by some writers (Obersteiner) the motor vago-glossopharyngeal nucleus. In the present case its cells, however, are perfectly healthy. By a process of exclusion, then, we are driven to localise the nucleus of origin of the motor fibres of the eleventh nerve in the sphere of the hypoglossal nucleus.

Anatomical evidence is hitherto wanting, but the clinical facts of nearly all cases of bulbar paralysis point to this conclusion.

Conclusions summarised.—1. Our conclusions with regard to the innervation of the facial muscles may be briefly summarised as follows, and are diagrammatically represented by Fig. 8.

The facial muscles may be divided into three divisions. First, the oculo-facial group, frontalis, orbicularis palpebrarum, and corrugator supercilii. Second, the middle group, elevators and depressors of the angle of the mouth, zygomatics, risorius, and buccinators. Third, the oro-facial group, or orbicularis oris. All these muscles are innervated by fibres included in the facial trunk, and are all paralysed, when the *nerve* is affected, as is shown by any ordinary case of Bell's paralysis. When, however, the facial nucleus only is diseased there results paralysis of the middle group only.[1] The upper group is paralysed when the oculo-motor nucleus is affected, the course of the fibres being probably the posterior longitudinal bundle.[2]

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[1] Kronthal, *op. cit.*

[2] Beevor and Horsley, *Proc. Roy. Soc.*, vol. xliv., 1888.

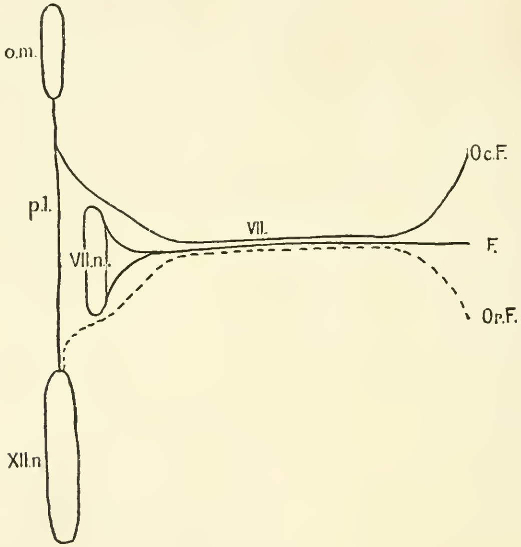

FIG. 8.

Diagram showing the origin and distribution of the fibres contained in the facial nerve trunk; o.m., oculo-motor nucleus including for diagrammatic purposes, the third, fourth, and sixth nuclei; p.l., posterior longitudinal bundle conveying fibres from the oculo-motor nucleus to Oc.F., the oculo-facial group; VII.n., the nucleus of the seventh nerve supplying fibres to F., the middle facial and lower muscles of expression; XII.n., the hypoglossal nucleus supplying fibres to Or.F., the oro-facial, or lip muscles represented by a broken line.

Paralysis of the orbicularis oris is associated with that of the hypoglossal group of muscles, and is therefore presumably innervated by that nucleus. The course of the fibres is at present obscure, but is possibly again the posterior longitudinal bundle.

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[1] Gowers, *Loc. cit.*

[2] We have reason to suspect that in total nuclear ophthalmoplegia this does occur. See also a case mentioned by Uhthoff, *Berl. Klin. Wochenschr.*, 1887, p. 913.

2. The eleventh nerve or accessorius vagi is known to contain motor fibres for the palate and vocal cords. The apparent nucleus of this nerve is indistinguishable from that of the vagus with which it is continuous. Its nerve roots may be regarded as the lowest fibres of the vagus, and its nucleus as the lowest part of the vagus nucleus. Its motor fibres which innervate the palate and larynx are, in all probability, derived from the region of the hypoglossal nucleus.

ON HEMISECTION OF THE SPINAL CORD.

BY WILLIAM ALDREN TURNER, M.B.EDIN., M.R.C.P.LOND.

SOME doubt has been recently[1] thrown upon the method of transmission of sensory impressions by the spinal cord, long ago enunciated by Brown-Séquard.[2] The conclusion, that all sensory fibres, with the exception of those subserving the muscular sense, decussated and passed up the opposite side, immediately or shortly after their entrance into the cord, was based on the records of clinical cases and on experiments made on guinea-pigs. Our intention here is to pass in review the whole series of recorded cases of "Brown-Séquard's paralysis" amounting to nearly 60 and to add a series of original experiments on monkeys, undertaken with the two-fold object; (1) to explain, if possible, the apparent discrepancies between the results obtained by the earlier observers and those more recently recorded; and (2) to ascertain the duration of the symptoms which might primarily result from hemisection of the spinal cord;—a point of very great importance in connection with the question of restitution of function.

The experiments were performed in the Neuro-Pathological laboratory of King's College. The facts of experiment here recorded were for the most part verified by Professor Ferrier.

1. *Clinical.*

It has been found necessary at the outset to divide the recorded cases of this paralysis into two great divisions; (1) those which were observed and recorded immediately after, or within a short time of the injury or onset of the disease; (2) Those which were not carefully observed until months or years after the onset of the symptoms. That this division has been rendered essential will be seen from what follows, but the statement may be made now, that the presence of a set of symptoms at any given time is no evidence of their existence either a short time previous or later on; a constant tendency to the restitution of the functions of the injured portion of the spinal cord occurring in those parts which remain intact.

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[1] Dr. F. W. Mott, *Proc. Physiol. Soc.*, January, 1891.

[2] Brown-Séquard, *Journal de la Physiologie*, vol. vi., and *Lancet* ii., 1868.

1. *Cases which were observed immediately after or within a short time of the onset of the symptoms.*[1]

Tactile sensibility.—In all the cases, which have been collected, the sense of touch was present on the paralysed side. In the great majority there was hyperæsthesia of this as well as of the other forms of sensation; in several it is simply stated that all forms of sensation were preserved; but in three (Nos. 9, 14, 22[2]) it is definitely noted that the sensibility to touch was normal, while pain and temperature were augmented. In the major number of the cases this state obtained both in the paralysed leg, as well as in the paralysed arm, when the lesion was situated in the upper cervical region. On the non-paralysed side or side opposite to the lesion one finds that in the greater number there is complete anæsthesia of all forms of sensation; in a few the sensibility is defective; in No. 39 (Gowers' case) there was no loss of tactile sensibility and only incomplete analgesia. In No. 42 although there was analgesia, the sensibility to touch was merely lessened; In 13, 55 and 56 tactile sensibility was only defective, while in 44 and 53 it was retained. In No. 52, the paralysed leg presented a hyperæsthesic condition, while the paralysed arm showed "loss of feeling"; the non-paralysed leg was anæsthetic, the corresponding arm being normal as regards both motion and sensation.

Sensibility to pain (pricking and pinching).—What has been said with regard to touch, holds good for pain on the paralysed side. As a rule there is hyperæsthesia of painful impressions. In No. 9, pain and temperature impressions were augmented, while touch was normal: in No. 14, pain only was increased. On the side opposite the lesion there is analgesia; in No. 13, there was analgesia and thermoanæsthesia, the sense of touch being only diminished: in No. 39, there was defective sense of pain without any affection of tactile sensibility; and in No. 56 with defective sense of touch and pain, there was loss of the sense of temperature. In No. 30, there was blunting of the sensibilities of pain and heat, the sense of touch being unaffected.

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[1] Cases of tumor of the spinal cord have, for obvious reasons, been left out of the collection.

[2] The numbers refer to the list of cases at the end of the paper.

Temperature sense.—This behaves in most cases as the other forms of sensibility, *i.e.*, increased on the paralysed side, and lost or diminished on the side opposite the lesion. But amongst the recorded cases there are several well-marked instances where this form of sensibility dissociated itself from the others. In Nos. 55 and 56 there was thermoanæsthesia on the non-paralysed side, existing with defective pain and tactile sensibilities. No. 55 is especially important, for the patient was seen two and a half years later, and although the other forms had to some extent been restored, there was still complete loss of temperature sense on the side opposite the lesion.

The sense of localisation.—Brown-Séquard's conception was that there existed perfect knowledge of the localisation of impressions on the paralysed and hyperæsthetic limb, while there was loss or great diminution of this faculty on the anæsthetic side. The collected cases appear to corroborate this view.

The muscular sense.—On this point there is more diversity of opinion, than on any one of those previously considered. Brown-Séquard believed that this sense (the sense of detecting difference of weights, or of telling the position of the limbs without the aid of sight) was in abeyance on the paralysed side; but Dr. Ferrier (BRAIN, vol. 7, p. 1) showed, as a result of hemisection of the spinal cord of a monkey, that the sense of position of the limb appeared absent, not on the paralysed but on the opposite side (viz. that on which there was cutaneous anæsthesia). In several of the collected cases no mention is made of this condition, while in others the only tests employed were those of "directing movements" or "perceiving strong pressure." It is therefore necessary, when noting the presence or absence of the muscular sense, to observe the sensibility of the skin to tactile impressions. In Nos. 11, 13, 15, 21, 53 and 57, the muscular sense was present on the apparently anæsthetic side, but on this side there was no loss of tactile sensibility. In Nos. 26 and 45 it was lost on the anæsthetic side, in the latter it is especially stated that the patient was unable to tell when the eyes were closed, the position of the toes, foot, or lower part of the leg, over which parts there was complete tactile anæsthesia. There was some uncertainty of the muscular sense on the paretic side in No. 54, but here the tactile sensibility was defective.

2. *The cases which were observed months or years after the onset of the symptoms.*

In a case recorded 3 months after the onset of the symptoms (No. 48) there was paralysis on the side of the lesion; and on the opposite side, defective sensibility to touch and tickling, with analgesia and thermoanæsthesia. In several seen six months after, both the amount of paralysis and the condition of the sensibility varied. In No. 40 there was on one side paresis with normal sensibility, and on the other anæsthesia of all forms of sensation. In No. 49, on the opposite side the sense of contact was defective, while that of pain and temperature were entirely lost. In No. 50 a weakness in the finer movements summed up the motor defect, while pain and temperature sensibilities were in complete abeyance on the opposite side. In No. 52, with paresis of arm and leg on one side, there was on the opposite side defective sensibility in the leg, while the arm was normal. In one seen twelve months after onset (No. 45) there existed paresis of one side with defective sensibilities on the other. In No. 55, which was seen two and a half years after the first observation, all forms of sensibility had returned in the leg with the exception of pain and temperature. No. 16 was seen three years after the onset and slight hyperæsthesia still existed on the paralysed side, and on the opposite side, although touch was present, there was analgesia. In two of Brown-Séquard's cases (Nos. 3 and 20) observed eleven and twenty-one years after a stab in the back, and a fall off a staircase respectively, hyperæsthesia existed on the paralysed side, which was also rigid, and on the other side all forms of sensation were abolished except tactile sensibility which was defective. On the other hand in a case seen twelve years after a similar injury (No. 53) on one side there was defective movement with normal sensibility, on the other touch, pain and temperature senses were only diminished.

It is necessary further to study with some detail those cases in which the lesion was situated in the cervical region of the spinal cord. Of the sixty collected cases, specific mention is made of the arms and legs in seventeen. Of these, thirteen show the usual appearance of "Brown-Séquard's paralysis" in both upper and lower limbs; the remaining four are of great interest when taken in conjunction with the experiments on monkeys shortly to be detailed. In case No. 3 (Brown-Séquard), as a result of a stab high up in the neck, a patient developed paralysis of the right arm and leg. On the paralysed leg there was hyperæsthesia of all forms of sensation, while on the non-paralysed there was defective tactile sensibility, analgesia and thermo-anæsthesia. On the right or paralysed hand, the two points of the æsthesiometer were recognised at 2-3 c.m., while the upper arm was anæsthetic, the senses of tickling, pain and temperature were exaggerated. On the non-paralysed limb there was more or less anæsthesia to all forms. In No. 6 (Perroud, quoted by Brown-Séquard) the "sense of contact" is stated to be diminished on the paralysed forearm, but no mention is made of the sensibility of the other arm. In No. 7 (Brown-Séquard), paralysis of the right arm and leg resulted from a stab in the neck. In the lower limbs there obtained paralysis with hyperæsthesia on one side, anæsthesia on the opposite side, while there was incomplete anæsthesia on both arms. Lastly in case 52 (Hoffman) the following condition was observed: Paralysis of the right leg with hyperæsthesia, paresis of the right arm with "loss of feeling"; anæsthesia of the right leg, while in the left arm "motion and sensation were good."

Among other facts in connection with the collected cases may be mentioned :—The organic functions are as a rule disturbed for a short period after the injury to the spinal cord, in the majority of cases incontinence of urine and fæces lasted from ten days to a fortnight. In a few cases constipation was present. The bilaterally associated movements of respiration are not affected, the two sides of the chest moving equally immediately after the lesion: In No. 52 paresis of the diaphragm on the side of the motor paralysis was noted, while in Nos. 46 and 54 special note is made of the fact that the abdominal muscles on the side of the lesion were paralysed. The condition of the reflexes calls for special mention. In nearly all the earlier cases recorded by Brown-Séquard and others, the superficial reflexes, especially the plantar, were observed to be augmented on the non-paralysed side, while in the later cases the condition of the knee jerk was noted. Immediately after the injury the knee jerk is absent or diminished on the paralysed side and normal on the opposite side. During the progress of the case, it returns and in course of time becomes somewhat exaggerated.

Before leaving the discussion of the recorded cases of this paralysis, it should be noted that only in a very few instances a *post-mortem* examination was obtained.[1]

It is, therefore, impossible to say whether in the majority of cases the lesion was a truly unilateral one or not. But arguing from the facts of experiments it appears probable that the major portion were of this one-sided character.

2. *Experimental.*

Before detailing the experiments, the methods of operation will be mentioned. Monkeys were used in every case, and the operation itself was regarded as a surgical procedure under strictly antiseptic precautions; in all cases the wound healed by the first intention. Chloroform was the anæsthetic employed. The vertebral spines and laminæ were exposed in the usual way, and the canal opened into with the bone forceps. The theca was incised mesially, and at either end of this incision two oblique cuts were made outwards, so as to expose the entrance of the posterior nerve roots on either side. The posterior median fissure is usually obscured by a blood-vessel. This having been moved to one or other side, a grooved transfixion knife was passed through the cord in the middle line of the body, and one-half of the cord severed. The wound was then closed.

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[1] These are Nos. 25, 27, 29, 39, and 46.

Experiments on animals made with a view to investigate conditions of sensation are beset with fallacies, and the symptoms may be differently interpreted. We have, therefore, in detailing the experiments, noted what we saw. The two chief difficulties which stand in the way of this method are—(1) Complications arising from reflex action. It is pointed out that this is excessive in the lower limb on the side opposite the lesion ; in some of the cases a drop of water or a breath of cold air on the non-paralysed foot setting up active movements of the leg. (2) As one can only infer the presence of sensation by the movements which occur, it is possible sensation may exist not evidenced by movements. In a few of the cases on the day following the operation, it was difficult to ascertain definitely the presence of a sense of touch owing to slight apathy, but after the first twenty-four or thirty hours the animal assumed its normal state.

EXPERIMENT 1.—*A section of the right side of the spinal cord at the level of the ninth dorsal nerve root* was made on April 11th, 1891. A well-conditioned male Rhœsus monkey was used. On the day following the operation, which is regarded as the *first* day of observation, and when all appearance of shock had passed off, the following condition was noted :—There was complete paralysis of the right lower limb, while the left, although it was freely moved when the animal lay upon its back and tried to escape, was not used for prehension when climbing the lattice-work of the cage. Its attention was at once arrested by ruffling the hair, pricking, pinching, and the application of a dull hot wire to the skin of the right lower limb, while it paid absolutely no heed to any of these forms of stimulation on the left. There was no reason to suppose from the manner in which it responded to the various forms of irritation that they were felt more keenly than normal. The right foot felt warmer than the left, while a surface thermometer recorded on the sole a difference of 16° F. between the two (right sole 86° F., left sole 70° F.). The right knee-jerk was not obtained, while the left was more marked than normal. The superficial reflexes, especially the plantar, were much more active on the left or non-paralysed limb. This condition remained until the *third day*, when, on touching the region of the left knee with the hot wire, the monkey would bring down its hand and scratch, but never struggle or show signs of discomfort. On the *fifth day* when pricked with a pin about the left knee, but much more evident when the hot wire was applied to the limb generally, it brought its hand down to the place touched and localised it. It took no notice of ruffing the hair on this side. The reaction to cutaneous irritation of the right side remained as before. On the *tenth day* the physical signs corresponded to those in the last note; it was evident that painful impressions were perceived, but much less readily than on the right limb. By the *twenty-fourth day* the power of flexing the right hip joint had returned. For the first time, the "clamp test" (Schiff) was introduced into the routine of testing, and this revealed interesting facts not altogether easy of interpretation. It was noted that when the clamp was applied to the right leg, struggling and other signs of discomfort immediately ensued, but there was no attempt to remove it, while if it was applied to the left leg, there was no sign of discomfort, but the leg was at once drawn up and the clamp was pulled off. It seemed as if the cause of this was the excessive reflex action of the non-paralysed side; to overcome this the legs were held, and the clamp again applied to the right leg; at once the struggling commenced, and continued until the clamp was withdrawn, while the monkey took absolutely no notice as long as the clamp was attached to the left side. On freeing the legs, however, the left was drawn up and the clamp pulled off. The temperature of the soles was the same. This doubtful condition remained for some days, but on the *thirty-eighth day* the following state was noted:—There is considerable return of the movements of the right leg, flexion and extension at the hip and knee joints being well effected. The left leg is now used much more extensively than before. The animal takes no notice of the pinching with forceps on the left side, but brings its hand down in an indefinite way when pricked gently at the root of the tail and the lower part of the back, and an equal degree of pricking causes more discomfort on the right than on the left side. When the animal's attention is diverted, ruffling the hair by blowing through a long glass tube invariably causes it to scratch. It does not take so much notice of the dull hot wire on the left as on the right side. The reactions to the clamp remain the same. On the *fifty-third day* the movements of the right leg had almost entirely returned, a slight drop-foot only indicating what had been a paralysed leg. There was also some wasting of the muscles of this limb. It took notice of pricking and pinching and ruffling the hair on the left leg as it did on the right. From this date onwards it was impossible to say which had been the anæsthetic limb; it responded as readily to all forms of stimulation on the one side as on the other.

On the *sixty-eighth day* (June 23rd, 1891) after the section of the right side of the cord, *a section of the left side at the level of the sixth dorsal nerve root was made.* The result of this was complete paralysis of both lower limbs, and complete anæsthesia of both limbs. It paid no attention to any forms of stimulation when applied to the legs, but readily when applied to the arms. The superficial reflexes on the right side now became markedly exaggerated. From this date onwards to the *one hundred and thirteenth day* after the original operation, when it was killed under an anæsthetic, the monkey showed no signs of returning motion or sensation. Both lower limbs became contracted and rigid and atrophied, the knee-jerks were active, the right remaining more marked than the left, and the superficial reflexes were also active.

Pathological examination.—At the seat of both incisions the dura mater was adherent to the surrounding bone, the membrane itself being slightly thickened. The spinal cord was removed and hardened in Müller's fluid. After some weeks, sections were made at the seat of both lesions, and double-stained with the Weigert-Pal and Carmine (Upson's) methods. In the first lesion, at the level of the ninth dorsal nerve, the following features were observed:—The incision was filled in by a layer of white fibrous tissue, which extended from the periphery to the line of the central canal. The whole of the right side of the cord had been destroyed, but in addition a small portion of the left postero-median column. The central grey matter on the left side maintained its normal shape, but its structure appeared slightly altered, owing probably to a certain amount of "traumatic degeneration," which had also increased the neuroglia of the white matter lying in close relation to it. At the sixth dorsal root, where the second hemisection was made, the right half of the spinal cord was intact, with the exception of the ascending degeneration, resulting from the lower lesion. The incision had not been filled in with new tissue, but remnants of very much degenerated cord substance were found in front and behind.

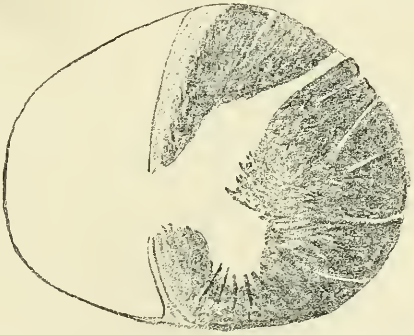

FIG. 1. (Exp. 1.) Representation of a section made through the lesion at the level of the ninth dorsal nerve roots. The unshaded portion of the section is the right side of the spinal cord. The anterior and posterior median septa are represented. The degeneration of the white matter on the left side of the cord is probably of "traumatic" origin.

EXPERIMENT 2.—*Section of the spinal cord on the left* *side at the level of the seventh dorsal nerve root* was made in a female Rhœsus monkey, June 10th, 1891. The animal recovered very rapidly from the operation, and presented the following features :—Complete paralysis and flaccidity of the left lower extremity, while the right was moved well in all directions; although, as occurred in the first experiment, the non-paralysed leg was not used for any useful purpose, as, for instance, when it was climbing the lattice-work of the cage. This was, no doubt, due to the fact that owing to the cutaneous anæsthesia, the sense of the position of the limb was lost, and, without the aid of vision, it did not know where its right leg was. When the skin of the left leg was tickled, scraped with a blunt instrument, or gently pricked and pinched, its hand was at once brought down accurately to the place which was irritated. Absolutely no notice was taken of any of those forms of irritation when applied to the right lower limb. The superficial reflexes on the right side were markedly exaggerated. The paralysed limb further communicated a feeling of greater warmth to the hand than the non-paralysed. By the *sixth day* the monkey localised correctly the places on the right leg where it was pricked, or when the skin was tickled by blowing upon it, but it never struggled or showed signs of discomfort. Similar reactions to the clamp test ensued in this case to those observed in the first experiment. When the clamp was applied to the left side, the animal invariably struggled, and appeared annoyed by the presence of the clamp, but never tried to remove it. On the right leg, however, it never showed the slightest sign of any discomfort, even when the clamp was kept applied for several minutes; it was only necessary to free the limb which was being held, when the leg was drawn up and the clamp removed.

During the succeeding weeks, when the animal was under observation, the responses to the tests showed little or no variation from what is stated above. All forms of irritation, including the clamp test, arrested its attention when applied to the left leg; on the right leg, however, although pricking, pinching, and the hot wire usually attracted notice, the response was obtuse, and a greater severity of stimulation was required. Return of movement was first noticed at the left hip-joint on the *twenty-second day*, and this proceeded until between the *fiftieth and sixtieth days;* movements had entirely returned, with the persistence, however, of slight drop-foot. The monkey was killed upon the one hundred and twenty-seventh day after the operation.

At the *autopsy*, the hemisection was found to be at the level of the seventh dorsal nerve root. After hardening the cord for some weeks in Müller's fluid, sections were cut and stained by the Weigert-Pal method. Sections made at the level of the lesion showed that the whole of the left half of the cord had been destroyed, and to some extent also the anterior and posterior median columns on the right side. The space intervening between the cut ends of the left side of the cord was filled with white fibrous tissue, in which there was no evidence of regenerating nerve fibres.

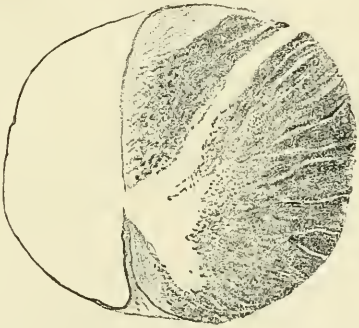

FIG. 2. (Exp. 2.) Section through the lesion at the level of the seventh dorsal nerve roots. The unshaded portion of the figure represents the left side of the spinal cord. The degeneration on the right side of the middle line is probably of "traumatic" nature.

These two experiments may be regarded as presenting the characteristic features of hemisection of the spinal cord in the mid-dorsal region. For a few days after the infliction of the injury, *all forms of sensation are lost on the side opposite the lesion*, while on the paralysed side all forms are preserved. After three to six days sensibility begins to return on the anæsthetic side. The first experiment showed that the order of return was sense of pain (heat) on the third day, sense of pain (pricking and pinching) on the fifth, while in the second all forms, pain (heat), pain (pricking and pinching) and touch had returned simultaneously by the sixth day. There exists some doubt concerning the late return of the sense of touch in the first experiment. Our notes indicate that for many days much dubiety existed regarding the sensibility of the non-paralysed leg, so that it is not improbable that the sense of touch returned considerably earlier than is noted here. A certain obtuseness of sensibility obtains for some time after its restitution; the monkey withstands severe forms of irritation on the side opposite the lesion.

In both the experiments, movement at the hip-joint on the paralysed side was observed between the twentieth and the twenty-fourth days; at the knee-joint between the thirtieth and fortieth days; while dorsiflexion of the ankle remained in abeyance for a long time.

EXPERIMENT 3.—*A section of the left side of the spinal cord of a male bonnet monkey* was made between *the third and fourth cervical nerves* on May 26th, 1891. On the day following the operation one observed, that there was complete paralysis of the left arm and leg; as regards the sensation, (1) of the lower limbs; the animal paid no attention when it was touched on either leg, but it was dull and apathetic; it however readily responded by bringing its hand down to the place when it was pricked, pinched or touched with a dull hot wire on the left leg. No such response was evoked when the right lower limb was similarly irritated. (2) Of the arms; all forms of irritation except ruffling the hair at once arrested its attention when applied to the right upper limb; but there was considerable difficulty in determining the exact condition of the left. It took no notice of ruffling the hair by blowing through a glass tube or of gently scraping the surface of the skin with a blunt instrument, while it would struggle slightly or open its eyes when this limb was pinched, pricked or touched with a hot wire. Owing to its apathetic state it was difficult to estimate the sensibility to touch on the extremities; the sensibility to pain and heat resolved itself as follows: analgesia of the non-paralysed leg, obtuseness of sensibility on the paralysed arm, while the paralysed leg and the non-paralysed arm presented normal reactions; there was no reason to suspect that there was any hyperæsthesia on these limbs. Tickling the sole of the right foot occasioned a lively reflex drawing up of the leg. The temperature of the left sole was 5° F. higher than the right. The right arm and leg were freely moved.

On the *seventh* day there was no sign of any return of movement in either limb on the left side, but when the hair on the right buttock and thigh was gently pulled or the skin lightly scraped or pricked, the animal at once brought down its right hand and scratched the place which was irritated. It was several times seen to spontaneously scratch the right thigh. And further, pricking the right foot and leg caused it to move away or, if held, to struggle. The same notice was taken when the left arm was pricked, but to a much less extent and the prick required to be more forcible. It was able to climb the lattice work entirely by means of its right arm and leg. On the *thirteenth* day, the first return of movement was noticed—flexion at the left hip joint; it responded to all forms of irritation (touch, pain, heat) in the left leg and the right arm and the right leg; much difficulty was experienced in ascertaining the sensibility of the left arm; the monkey was never aroused from its occupation when the arm was touched, but when pricked or when the hair was pulled it looked up but did not show signs of discomfort. On the *twenty-ninth* day the movements of the left leg were well marked at the hip and knee joints; the monkey readily took notice of all forms of stimulations on the legs. As in the first experiment the clamp was removed from the right leg, but never from the left. It was still difficult to determine the tactile sensibility of the left arm, it responded to painful impressions, but less readily than on the right side. By the *fifty-fifth* day some power of movement over the shoulder joint had been regained, while the increase in motor power of the left leg had advanced. There seemed to be no doubt that touching the left arm and hand or very gently pulling the hairs were felt, because the animal invariably looked round and moved away, when this was done. Its perception of tactile impressions on this limb was distinctly blunted, as also was its perception of painful impressions. On the *one hundred and sixth day* the note was made that there was almost complete return of the movements of the left leg; the left arm was still paretic; it could not raise the elbow higher than the shoulder and there was marked wrist-drop. The sensibility remained the same as at the last note. The monkey died on the one hundred and twenty-sixth day after the operation.

Pathological examination.—Owing to a considerable amount of adhesion between the spinal cord, dura mater and vertebral column in the upper cervical region, some difficulty was experienced in removing the cord, with the result that the latter was torn at the seat of the lesion, so that we are unable to figure the exact amount of destruction of the cord. Careful examination has been made of the ascending and descending degenerations. The tracts which degenerate upwards are, on the left side, the whole of Goll's column, the direct cerebellar tract, and the ascending antero-lateral tract of Gowers. On the right side of the posterior median fissure there is a small linear area of degeneration in the postero-median column. The descending degenerations are the crossed and direct pyramidal tracts on the left side, and

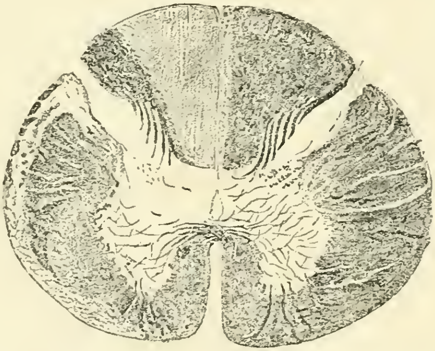

FIG. 3. (Exp. 3.) Transverse section of the spinal cord at the level of the second cervical nerve root, showing ascending degenerations following a section of the left side of the cord between the third and fourth cervical nerves.

a small portion of the direct pyramidal tract on the right side. The lesion itself was situated between the third and fourth cervical nerves on the left side.

EXPERIMENT 4.—*A section of the left side of the spinal cord at the level of the third cervical nerve* was made on Nov. 3rd, 1891. A large male Rhœsus monkey was used. On the day after the operation the state of motion and sensation was as follows: The left arm and leg were completely paralysed, while the right were freely moved at all the joints. All forms of cutaneous irritation applied to the left leg, buttock and flank (gently ruffling the hair, scraping the surface of the skin, pulling the hair, pricking, pinching, and the dull hot wire) at once arrested the animal's attention. When the clamp was applied to the sole of the left foot or to the skin of the leg, struggling at once ensued, but there was never any attempt to remove it, and this continued as long as the clamp remained attached. Absolutely no notice was taken of any of these forms of irritation on the right leg, buttock or flank, but when the clamp was attached to the right sole, the leg (unless held) was speedily drawn up and the clamp pulled off. Pricking, pinching, and the clamp forceps gave rise only to slight signs of discomfort when applied to the right upper limb, while the monkey struggled considerably when the left arm was pricked or pinched or touched with

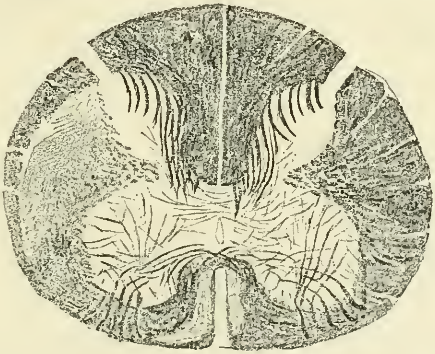

FIG. 4. (Exp. 3.) Transverse section of the spinal cord at the level of the seventh cervical nerve, showing degenerations descending from a section of the left side between the third and fourth cervical nerves.

the hot wire. It paid no heed to gently scraping the skin or ruffling the hair of this limb, although its attention was at once arrested when a drop of water was allowed to fall upon either hand. It struggled when the clamp was applied to both hands, but less on the right than on the left side. It never tried to remove the forceps from the left hand. The right knee jerk was well marked, the left was present but feeble. The superficial reflexes on the right side were very active.

On the *second* day the monkey again removed the clamp from the right sole, but there was no evidence of any discomfort when it was applied. It never attempted to remove the clamp from the left sole, although it struggled as long as the pressure of the clamp was kept up. In all other respects, it remained as on the previous day.

On the *fourth* day, the reactions were as at the previous observations, but with this addition that pricking the right leg or right root of the tail would occasion a slight amount of struggling, as if there were some, but defective, perception of painful impression.

On the *fifth* day the monkey died.

The autopsy revealed a hemisection of the spinal cord at the level of the third cervical nerve.

Experiment 5.—*Section of the left side of the spinal cord at the level of the fourth cervical nerve.* The section was made on November 17th, 1891.

On the following day there was paralysis of the left leg and almost complete paralysis of the left arm, a slight movement at the shoulder joint being occasionally observed. Absolutely no notice was taken of any form of irritation applied to the right lower limb; when the clamp forceps was applied to the sole of the foot or elsewhere on the leg, a sudden spasmodic movement ensued of probably reflex nature, but the clamp was never removed, nor was there any attempt at removal. Similar forms of irritation on the left lower limb caused struggling and attempts to move away; and on the application of the forceps evident signs of discomfort, but no endeavour to remove it. The monkey bore, with only a small amount of struggling, pricking and pinching the right upper limb, but at once responded to a touch either of a blunt instrument or of a drop of water let fall upon its hand. It at once took notice of pricking and pinching the left upper limb and also when the clamp was applied, but we were unable to detect at first any evidence of a sense of contact on this limb, as the monkey paid no attention to ruffling the hair or dropping water on its left hand. The superficial reflexes of the right lower limb were very marked, and the right knee jerk was readily obtained. The left knee jerk although present was feeble. The temperature of the right sole was 86° F, while the left was 94° F, that of the right palm was 84° F, while the left was 94° F. On the *second* day similar tests were employed, but no alteration was noted in the condition of sensation ; special attention was devoted to the tactile sensibility of the left hand, but no evidence of any recognition of tactile impression was obtained. On the *third* day, however, when the monkey was lying quietly before the fire, a drop of water let fall upon its left hand at once arrested its attention. The right leg remained completely anæsthetic to all forms of irritation. There was no sign of discomfort when the clamp was applied to the limb, nor was there any attempt to remove it. The right arm and left leg remained as before This condition was confirmed daily until the *eighth* day, when on applying the forceps to the left arm, the monkey brought round its right hand and tried to remove them, and, further, when water was dropped upon the left hand or when the arm was gently scraped, it looked towards its hand, and attempted to move away from the irritation. This was the first indication of something more than a mere feeling of contact on the paralysed arm. The right leg remained completely anæsthetic to all forms of irritation. There was some increase in the amount of movement noted on the first day, but the animal was yet unable to stand or sit up. On the *sixteenth day* the right leg remained completely anæsthetic to all stimulation, excepting that when pricked at the root of the tail on the right side, and when the skin of this region was blown upon, the monkey brought its hand round to the place and scratched it.

There was no attempt to remove the forceps from the right foot; although it could sit up, it was unable to progress. It at once responded when touched on the arms and the left leg: pricking the right arm caused less evidence of discomfort than when the left arm and leg were similarly irritated. On the *twentieth day* the condition remained as recorded in the last note.

The three experiments just recorded of hemisection in the upper cervical region present some variations in the amount of sensory disturbance. The condition obtained in the third experiment was opposed to previously recorded facts, and as the monkey was a troublesome one to test, the other two experiments (Nos. 4 and 5) were performed. These latter presented symptoms more in harmony with what occurred after hemisection in the dorsal region. On the arm opposite the lesion, the sense of pain was abolished or defective, but contact was retained. On the paralysed arm all forms of sensibility (pain and contact) were present, at any rate within a short time after the operation. These experiments also indicate variations in the duration of the symptoms and in the occurrence of restitution. In the third experiment sensibility had returned in the lower limb opposite the lesion by the seventh day after the operation. In the fourth experiment evidence of the return of painful sensibility was obtained on the fourth day. In the fifth experiment the lower limb opposite the lesion remained completely anæsthetic to all forms of irritation up till the twentieth day. The sense of pain on the non-paralysed arm remained very defective.

The Sensory Disturbances.

These experiments confirm the theory, which was originally advanced by Brown-Séquard, that the sensory fibres decussate immediately after, or within a short distance of, their entrance into the spinal cord and pass up the opposite side, while the motor fibres pass down the same side of the cord. This, however, appears to be only true of the sensory fibres for the lower, or hinder extremities. As far as our experiments show, a modification of this arrangement is necessary for the upper or fore-limbs. That complete decussation of the sensory fibres from the arms occurs is shown by the facts of cerebral hemianæsthesia, but at the present time it appears doubtful where this crossing takes place. It is evident from the experiments which have been recorded, that monkeys vary both in the amount of sensory disturbance which follows hemisection and in the duration of the symptoms before restitution occurs; and the recorded cases in man of lesion in the upper cervical region show some irregularity in the distribution of the sensory defects in the arms. The facts of the experiments in the upper cervical region show that all forms of sensibility were not abolished on the non-paralysed arm; sensibility to painful impressions was absent or defective, except in the third experiment, while the sense of contact was retained. On the paralysed arm, with retention of the sense of pain, evidence of tactile sensibility was obtained within a short period of the operation. This would appear to indicate that the fibres subserving tactile sensibility for the arms pass up both sides of the cervical cord, while those subserving painful and temperature sensibilities mainly pass up the opposite side. On the other hand, the experiments indicate that all forms of sensibility, with the exception of a sense of pressure in some monkeys, notably in experiment 4, are abolished in the lower limbs, temporarily at least, on the side opposite the lesion. In this connection we will here refer to the existence of a sense of pressure, more or less accurately localised, as shown by the clamp test. When the clamp was applied to the skin of the foot on the non-paralysed or anæsthetic side, the leg was at once drawn up and the clamp removed, while the monkey never exhibited any signs of discomfort. When, on the other hand, the clamp was applied to the paralysed foot, the animal at once began to struggle and bite, and show signs of annoyance, which continued during the time the clamp remained on, but it never tried to remove it. If, however, the anæsthetic limb was held, the animal never attempted to remove the clamp until the limb was set free and its movements unrestrained. In the fifth experiment, as long as the limb remained anæsthetic to all other forms of stimulation, no attempt was ever made to remove the clamp forceps.

There is, perhaps, greater difficulty in determining the condition of the muscular sense in animals, the conclusions depending entirely on the interpretation of the observed facts. The following observations appear to indicate that there is loss of the sense of movement in the lower limb opposite the lesion. When the animal was lying on its back, it could freely move and kick with the anæsthetic limb, but this leg was never used for prehension, as, for instance, when it was climbing the lattice-work of the cage. If a finger or other object was inserted between the hallux and the toes, no attempt was made to grasp it. If, when the eyes were blindfolded, the leg was moved about, the monkey never offered any resistance. If by chance the leg was caught upon an obstacle lying upon the floor, it was quite incapable of extricating it. All this, however, changed when the cutaneous sensibility returned.

The Restitution of Function.

1. *The motor functions.*—The collected cases and the experiments detailed above show that the functions of the side of the spinal cord which has been injured are in course of time restored. It has been pointed out that the movements returned in the paralysed limbs at different periods after the operation. The first movement to return is flexion at the hip-joint, and this occurred from a fortnight to three weeks after the infliction of the injury. Closely associated with this is the counter-movement of extension, and a short time later movements at the knee-joints are regained. Drop-foot remains for a very long time, apparently, after some of the movements of the hallux and toes have returned. Return of the functions of the paralysed arm occur much later than in the paralysed leg. First, movement at the shoulder joint, followed a little later by flexion and extension at the elbow ; in the third experiment, drop-wrist and inability to move the thumb and fingers persisted one hundred and twenty days after the operation. It therefore comes to be a very important point to ascertain

how this restoration takes place. Regeneration of the white fibres of the spinal cord probably does not occur, at any rate, in the higher mammals, although definite evidence on this point is not yet forthcoming. Eichhorst (*Virchow's Archiv.*, 1874, p. 1) found new nerve elements in the scar tissue resulting from section of the spinal cord in young dogs and rabbits. Our own observations do not confirm this, for the cicatricial tissue which filled in the gap made by the knife at the time of the operation in no case presented any evidence of regenerating nerve fibres. As no regeneration took place at the seat of the lesion in the experiments under discussion, the impressions were probably conveyed to the parts below the lesion by the opening up of decussating paths from the opposite uninjured side. Experiments seem to corroborate this view. The first experiment demonstrated that after the recovery of movement from a section of one side of the cord, section of the opposite side a little higher up caused permanent paralysis of both lower limbs. And two experiments performed by Rossolymo (*Neurol, Centralbl.*, 1887, p. 292) on guinea-pigs, entirely confirm this. In one experiment he performed a hemisection of the spinal cord at the tenth dorsal nerve root; and when the paralysis of the hind limb resulting from this had passed off, he made *on the same side* another section just behind the decussation of the pyramids. This occasioned paralysis of the fore limb on the same side, while the hind limb escaped. In a second experiment, after restoration of the movement resulting from a hemisection in the lower dorsal region, he made a vertical longitudinal incision throughout the lumbar enlargement, with the result that the leg was a second time paralysed. He had previously shown that such an incision produced no paralytic effect on either limb. Dr. Mott's experiments (*Proc. of Physiol. Soc. in Journal of Physiology*, Jan., 1891) on stimulating the motor cortex after recovery from the paralysis of a hemisection confirm this, for he found that excitation of the cortical area of the opposite leg evoked movements in both legs.

2. *Sensory functions.*—The restoration of these functions is a subject which has not as yet received very much atten-

tion. In cases of "Brown-Séquard's paralysis" which have been observed several months or years after the onset of the symptoms, no very marked improvement is found to have occurred in the sensory conditions: in one example seen twenty one years after the fall which caused the paralysis, all forms of sensation were abolished with the exception of touch, which was very defective. An analysis of the order and date of return of the sensibilities in the monkeys, which have been the subjects of our experiments, shows a condition differing considerably from the recorded cases of disease or injury in Man. In three of the experiments evidence of the return of the perception of painful impressions was obtained by the fifth or seventh day after the operation. The perception of these impressions was at first very defective. An amount of pricking and heat which caused distinct manifestations of feeling on the paralysed limb, on the non-paralysed only provoked a sense of slight discomfort; the monkey scratched the place irritated, but never or rarely struggled. In the second and third experiments the perception of tactile impressions returned contemporaneously with that of pain. It appears that in the monkey at any rate, probably owing to the less highly organised state of its spinal cord restitution of the sensory functions occurs at a much earlier date after the infliction of an injury, than obtains in the highly organised cord of man.

The Paths of Sensation.

Brown-Séquard's view of the existence of special conductors for the diverse kinds of sensory impressions and of different paths for their conduction in the cord, was based on the facts of many of the cases previously alluded to, viz., that one kind of sensibility was preserved while another was lost. He concluded that the temperature sense was probably conducted by the central grey matter, painful impressions by the posterior and lateral parts of the grey matter, while the tactile sense was conducted by the anterior parts of the cord. More recent observers have suggested other paths for the conduction of sensory impressions. Schiff suggested that tactile sensibility probably passed by the posterior columns, painful impressions being conveyed by the grey matter, while Dr. Gowers has advocated the antero-lateral ascending tract as a probable path of painful sensations. Experiments have been performed recently which do not corroborate these views.[1] Section of the outer portion of the lateral column in a monkey produced no effect upon sensibility below the lesion, while section of the whole of one side of the spinal cord, with the exception of the anterior and posterior median columns, gave rise to total anæsthesia and analgesia on the opposite side. The experiments tend to show that all forms of sensation pass up the cord in very close relation to each other, and their synchronous restoration after hemisection would favour this view. It has been suggested, because the ascending degenerations which follow hemisection are limited to the same side of the cord, that therefore the sensory fibres do not entirely decussate. It is true that anatomically the ascending degenerations are limited to the same side as the lesion, but clinically the anæsthesia is on the opposite side. The recent experimental work of Messrs. Gotch and Horsley[2] on the electro-motive conductivity of the spinal cord, favours the views of Longet and Schiff, that the posterior columns on the same side are the main afferent channels. Their method of research undoubtedly shows the existence of unbroken tracts of fibres passing up the cord on the same side as the entering posterior nerve roots, which the degenerative method also brings into prominence, but neither method distinguishes between centripetal impressions and sensation proper. Our experiments indicate that the tracts subserving sensation, until restitution occurs, pass up the opposite side of the spinal cord.

The reflexes.—The state of the reflexes, both superficial and deep, is of some clinical interest.

The experiments show that immediately after, and for a short time subsequently, the knee-jerk on the side of the lesion was in abeyance or very much diminished, while that in the opposite side remained normal. Within a short time (a few days in most cases) the hitherto absent knee-jerk returned, at first sluggishly, and then of normal strength. In the first experiment, where resection of the opposite side of the cord was performed, the knee-jerks showed similar variations, absent on the side of the lesion, of apparently normal amount on the opposite side. As rigidity and contracture proceeded, both knee-jerks became exaggerated; the superficial reflexes, especially that occasioned by tickling the sole of the foot, were in all cases found to be intensely exaggerated on the side opposite the lesion, those on the same side were either in abeyance or only slightly marked. The exaggeration of these reflexes remained for some weeks, when they gradually diminished to the normal extent. In the first experiment, where a second section was made on the opposite side, similar exaggerations occurred on the side opposite to the lesion; they showed no signs of diminution, while those on the other side increased as rigidity and contracture progressed.

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[1] Ferrier, "Croonian Lectures," 1890, p. 95.

[2] Gotch and Horsley, "Croonian Lectures," 1891. *Phil. Trans.*, vol. 182B., pp. 267-526.

Vaso-motor and trophic effects.—Immediately following the operation, the temperature on the paralysed side was found to be considerably higher than on the non-paralysed. This lasted for several days, but with the return of the motor functions, the elevation of the temperature subsided. In one experiment the temperature on both sides had become equal by the twenty-fifth day.

The muscles of the paralysed limb atrophy to a small extent, the circumference of the affected limb was less, and the muscles were soft and flabby. Their reaction to electricity was not materially altered. The limb remained slightly atrophied after the return of movement. Cutaneous tropho-neuroses were observed in only one case on the side opposite the lesion.

Organic functions.—In the recorded cases of wound of the spinal cord giving rise to the symptoms and signs of a half lesion, involuntary passage of urine and fæces was noted for some days. This was never noticed in any of our experiments, although retention of urine was usually present for twenty-four hours after the operation.

In none of the experiments was any impairment of the respiration observed, not even in the high hemisections immediately after the operation.

Addendum.—The following is the condition of the monkey illustrating the fifth experiment on the *thirtieth day* after the operation :—There is perception and localisation of tactile and painful impressions on the left upper and lower extremities, and the left side of the trunk. On the right upper limb the sense of touch is well marked, but the sense of pain is defective. On the right lower limb tactile and painful impressions are localised above the knee, while below the knee anæsthesia of both forms remains. There is rigidity and contracture of the paralysed extremities, while there is very little evidence of return of movement even at the left hip joint.

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ON THREE CASES OF HYSTERIA IN MEN.

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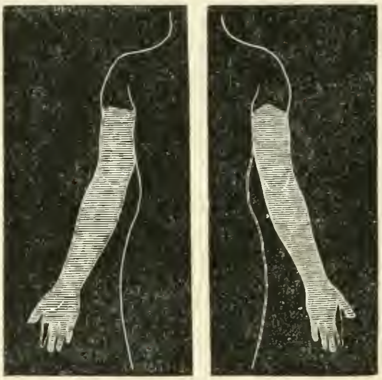
I WILL first of all give an account of the cases.

CASE I.—Stephen M., æt. thirty-three, a mattress maker. His family history was unimportant; his parents were healthy, but his mother was "nervous." No one in the family had ever had fits. At the age of ten months the patient had an attack of infantile paralysis, and since that time his right leg has been wasted; when seventeen years old he had inflammation of the lungs, and at twenty-nine an attack of "measles," during which he was for some days very ill. Twelve months ago he had an attack of "nervousness" and a troublesome cough. He is married and has one healthy child. Careful enquiry elicited no history of syphilis. He used to be a heavy drinker, but lately has taken less, though he is still probably intemperate, acknowledging to three or four pints of beer daily. He has never worked in lead.

During the last few months he has suffered from attacks of vertigo whilst walking; they last for ten minutes, occur about once in two or three days, and during them he has to stop and hold on to the railings or any convenient support. They begin with a feeling of numbness, starting from the feet and passing up through the body to the head, together with singing noises in the ears, severe pain in the forehead, and throbbing of the temples. There is no globus. His sight is not affected, he never falls, nor loses consciousness; only once "everything seemed to be going round," but on all other occasions this was not the case. He now complains of numbness in the fingers of the right hand.

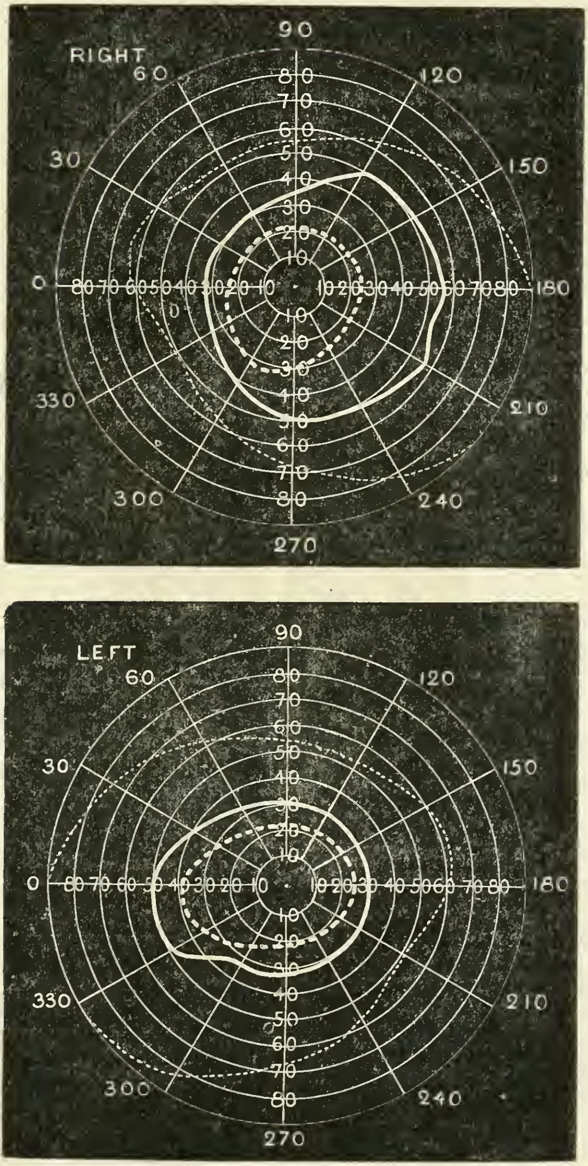
A week ago he had a pain in the left arm, and three days later, whilst at work cutting out the stuff of which the mattresses are made, his right hand and arm became numb, and he lost power in them: this numbness was unaccompanied by tingling or pain. Since then he has been unable to open his fingers, and has been obliged to give up his work. He is ambi-dextrous. In addition he suffers from attacks of morning sickness, from headaches, and his legs often feel very weak and powerless. He is giddy, especially in the dark, and has had a trembling of the hands for some time. There is no interference with micturition or defæcation.



Case I.

The shading indicates loss of sensation.

The patient was an anæmic man, fairly well-built. Examination of the thoracic and abdominal organs detected nothing abnormal. His pulse was regular, his tongue moist and thickly furred. The muscular system was well developed, except in the right lower extremity; in this the anterior muscles of the thigh were small and wasted, the right thigh measuring only sixteen inches in circumference, whilst the left measured twenty inches. The muscles of the leg showed a less degree of wasting, and the whole limb was a little shorter than the other. The right knee jerk was absent: sensation was unaffected. This condition of the right leg the patient stated had existed from



Case I.

The inner thick dotted line indicates the field for red.
The outer thick white line indicates the field for white.

infancy, and was doubtless the result of the attack of infantile paralysis above mentioned. There was a very rapid, constant, fine tremor of the hands, which was not increased by movement, and from its characters might reasonably be attributed to alcohol.

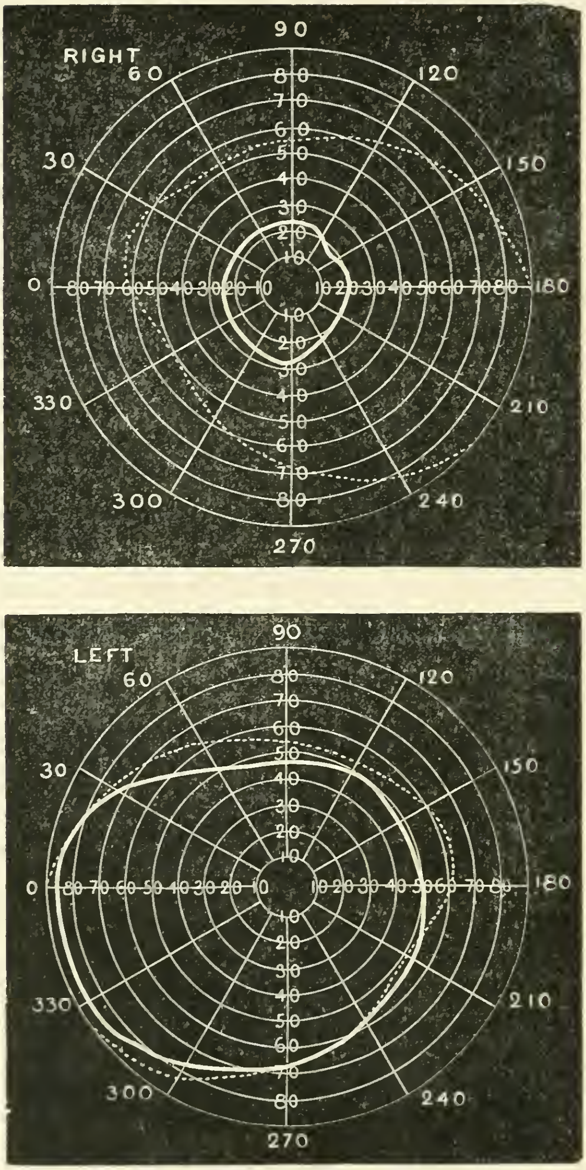
The dynamometer gave 21 kilos for the right, 38 for the left hand.

The right wrist and fingers were kept in a position of partial flexion, and there was a general loss of power, accompanied by some inco-ordination in all the movements of the right forearm, hand and fingers. No movements were entirely absent, and the paresis was most marked in the long extensors of the wrist and fingers, and in the action of abduction of the fingers from the middle line performed by the interossei. The other muscles, though weak, were affected in less degree, the movements of the thumb being least involved. The shoulder muscles acted normally. There was not the least evidence that the paresis could be attributed to lead. Sensation was almost lost to touch, pain, and temperature in the right upper extremity over the area shaded in the figure. Over the index finger, however, sensation was normal. The muscular sense was also completely lost in the fingers, and the patient could detect no difference between different weights ; in the index finger this was not the case, and he could accurately distinguish between various coins by their weight ; moreover the distance at which the points of the æsthesiometer could be detected as two was in this finger the same as over the fingers of the left hand. There was partial loss of muscular sense in the forearm. Elsewhere over the body sensation was entirely unaffected, and there were no tender spots. The sense of smell was normal ; taste was very deficient on the right side of the tongue, pepper and quinine being indistinguishable. The bilateral contraction of the visual fields is indicated by the charts. The left knee jerk and superficial reflexes were normal. There was no paralysis of any cranial nerve. The pupils were equal, and reacted well to light and accommodation. It should have been mentioned above that the right forearm and hand were colder than the left, and slightly livid, and that the electrical reactions of the muscles were normal.

Under general treatment with faradization and rubbing of the affected limb he gradually improved, and at the end of four weeks was able to return to work with fair power in the right hand, and with marked improvement of sensation. At the end of two months he considered himself well, though sensation was still a little less acute over the right than over the left hand, and he ceased to attend.

Case II.—The second patient, Thos. S., æt. twenty-eight, came to me in December, 1890, complaining of "fits." He was a quarryman by occupation, and was a strong, healthy-looking man of about middle size. He was somewhat excitable in his manner of speaking; intelligence good. The family history was good: both parents were healthy, his father being a temperate man of mild disposition; he had ten brothers and sisters, all of whom were healthy. None of the family had ever suffered from convulsions or fits of any kind, nor from chorea, insanity, or asthma. He was a married man, and the father of five healthy children. He had always been a teetotaller, and had never had any bad illness. He was of a very religious disposition, and was fond of taking part in religious revival services. He had not been engaged in any excitement of this kind before the onset of the first fit, which came on without apparent cause, whilst at his usual work in a quarry during very cold weather four years ago. Since then he had had a great many attacks, for none of which he could trace a cause, except that either very hot or very cold weather always seemed to bring them on. They generally occurred in the evening or at night. Sometimes he would go for some months without an attack, and then would have a number of them in a short time.

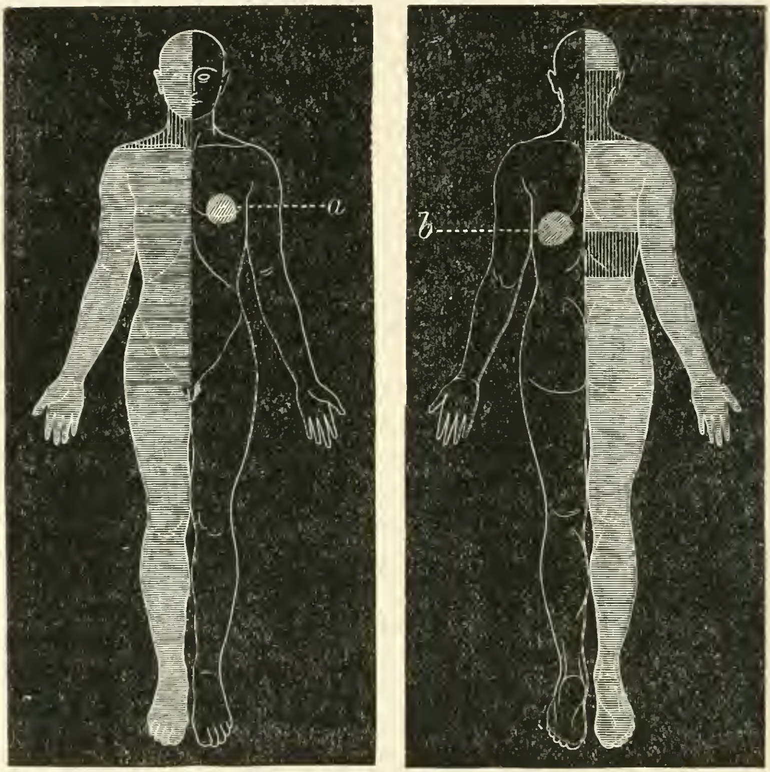
He gave the following description of the fits:—There is first a throbbing in the temples, which lasts from three to five minutes, and as it passes off a feeling of "deadness," numbness, or loss of sensation comes on in the tips of the fingers, and passes up through the arms to the back of the head, in which he at once has a severe pain as if he had received a



Case II.

The thick white line indicates the field for white.

violent blow there. This is followed by pains in the forehead; at the same time the numbness spreads from the shoulders all over the body, but seems especially to pass into the pit of the stomach and the left side of the chest. These symptoms last from a few seconds to a minute. He does not generally fall, because he sits down at once when he feels it coming on. During this part of the attack his feet are agitated, and beat against the floor. The numbness is succeeded by tingling, which he compares to the pricking produced by a faradic current (with which he had at one time been treated). This tingling begins in the fingers, and spreads all over the body as the numbness did, whilst the pains in the head become more severe. At the moment when the numbness dies away he feels faint, as if sinking through the earth. Sometimes there is loss of consciousness. More often there is not, but a condition in which he is unable to speak but hears everything that is said around him. Loss of consciousness appears to come on only when the pains in the head are of exceptional intensity. This faintness may last half an hour, or less. He has occasionally fallen in it, but has never sustained any greater injury than a trifling bruise from the fall. In the faintness he struggles "as if fighting against a battery," throws his arms and legs about and grinds his teeth. He does not scream, but moans and makes "gurgling" noises in his throat. He is always better if left alone, and if held his struggles become more violent. Occasionally, and especially when he is coming round from those attacks in which there has been brief loss of consciousness, he talks wildly, chiefly about religious matters or political questions, and then his prevailing idea is one of intense anxiety as to the state of his neighbours' souls. There are apparently no hallucinations, but when he opens his eyes at the end of the attack everything appears to be coloured, sometimes with all the colours of the rainbow, at others of a uniform red or blue tint. The attack ends with violent palpitation, which prevents sleep, although he feels intensely drowsy. There is no feeling of weakness or exhaustion after them. Palpitation never occurs except at the finish, and throughout there is no globus nor are there noises in the ears. The fits rarely occur singly, but in series of three or four. The greatest number at a time was five, and the whole series then lasted three-quarters of an hour. He has never bitten his tongue nor passed water in any of them.



CASE II.

Transverse shading—Anæsthetic area.

Vertical shading—Areas unaffected, or only very slight loss of sensation.

a. Hysterogenic area.

Transverse shading—Anæsthesia and analgesia.

Vertical shading—A slighter degree of anæsthesia.

b. Area of hyperæsthesia.

Apart from the attacks he often suffers from severe pains in the back of the head, but otherwise enjoys good health. He sleeps well, and his appetite is good. He suffers from constipation.

Examination showed that there was no paralysis of any muscle; the muscles were generally well developed, firm, and of normal strength. The knee jerks were present, the plantar reflexes absent; other superficial and deep reflexes normal. Physical signs in thorax and abdomen normal. Pupils equal, react well, and are slightly dilated; optic discs and fundus healthy.

There was well-marked anæsthesia over the right side of the body, which was accurately limited by the middle line in front and behind. Anæsthesia was complete to pain, a prick not being felt, almost complete to touch and to temperature. This anæsthesia was less marked on the neck, especially on the back of it, and over an area on the right lower ribs posteriorly, which is indicated on the chart. The right conjunctiva was also normal in respect to sensation, but the left conjunctiva was anæsthetic. The right side of the tongue and buccal mucous membrane were also insensitive. At the lower angle of the left scapula was an area of hyperæsthesia, and another about the size of a five shilling piece just below the left nipple. Irritation of this latter area produced feelings of numbness and tingling, and on continuing the irritation he lay back on the sofa, with eyes turned up under nearly closed eyelids, the limbs became momentarily rigid, and were subsequently agitated by slight clonic spasms, and there appeared to be partial loss of consciousness, the whole attack lasting about a minute or so.

In addition to the other symptoms, loss of taste was detected on the right side of the tongue (quinine and constant current) and of smell in the right nostril (asafœtida, creosote, and ammonia); there was well-marked contraction of the right visual field. Under treatment he improved, and the fits ceased, but as he was only six weeks under observation, it could not be said with certainty that the improvement was permanent. He told us that attempts at hypnotism had formerly been made upon him, but always unsuccessfully.

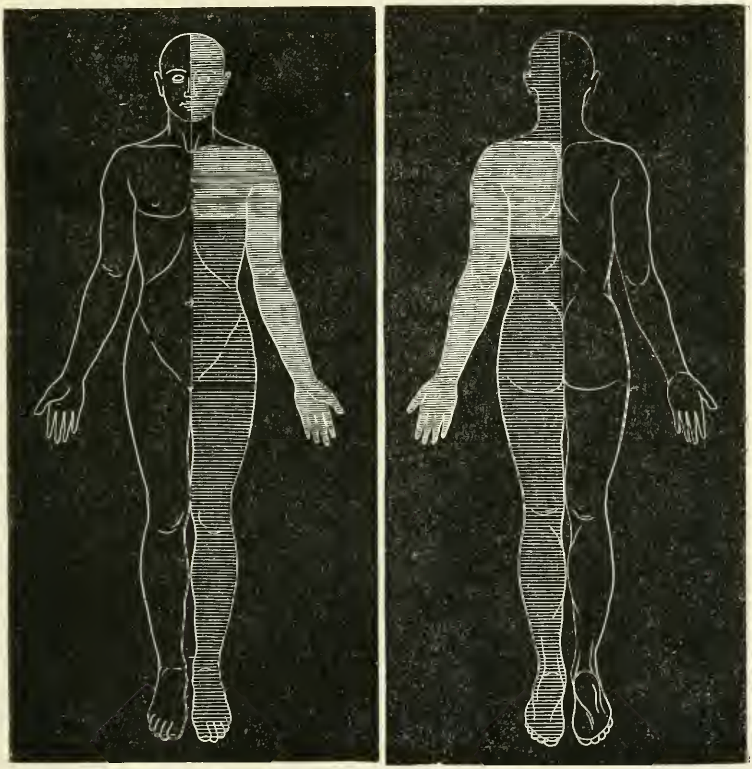
Case III.—John J., æt. forty-two, a cabman, complained of pain, stiffness and contraction in the left arm and elbow. On enquiry there was a history of gout in the family, an uncle having died of it; his father died of a malignant tumour of the neck, and his mother of inflammation of the bowels. He is an only child. No relative, so far as he knew, had ever been insane or suffered from fits.

He had himself always been healthy, had never had rheumatic fever nor gout, nor convulsions of any kind. There was no history of syphilis ; he had not been a very heavy drinker, although not strictly temperate in his habits.

The present illness began twelve months ago, with pain in the left arm, which came on during one night ; the pain gradually grew worse, and four or five months ago passed down to the elbow, shooting from the elbow over the back of the forearm and hand into the tips of the fingers. The pains seem to start from a tender spot just above the internal condyle of the humerus, on the posterior aspect of the arm, and to shoot thence into the hand and shoulder. Nearly four months ago he was thrown out of a trap, and fell on his head, not striking his arm at all ; he was rather shaken by the fall, and remained in hospital for two weeks, at the end of which time he was discharged, apparently well. Three months ago, or shortly after leaving the hospital, he found the elbow becoming stiff, and that movements were attended with pain in it ; this condition of painful stiffness has gradually increased until at present he is unable to bend it at all.

As to general symptoms, his appetite is poor, he is very sleepy in the daytime, and is thirsty, drinking a great deal of cold water. His bowels are regular. When in the hospital after the accident he suffered for a short time from inability to pass water ; this soon passed away, but since that time he has had to get up two or three times at night to micturate. Examination of the urine showed that it contained a deposit of urates, but no sugar nor albumen. His tongue was dry and thickly furred ; he had, however, no dyspeptic symptoms. The heart, lungs and abdominal organs were normal, and his pulse regular and strong :—68 to the minute. His face was flushed, but otherwise he looked healthy.

The left arm was kept flexed at the elbow, so that the forearm was at right angles to the arm and supinated. This position of the forearm was due to a state of rigid spasm in the biceps. By continued pressure the spasm could be overcome and the arm nearly but not quite straightened, and during this forcible extension the patient complained of much pain in the elbow. He was entirely unable to extend the forearm himself. The elbow-joint itself was perfectly healthy. There was a tender spot behind and above the internal condyle, and also in front of the elbow on the outer side. There was no paralysis of any muscles, which were generally well developed, firm, resistant and not flabby. Electrical reactions normal. The knee jerks were both present, the left a little more active than the right, and



Case III.

Over the area marked by the widely separated lines sensation was deficient, as compared with the right side.

Over the more deeply shaded parts there was more decided anæsthesia.

the superficial reflexes were normal. The contraction of the left arm placed it exactly in the position in which he was accustomed to maintain it in holding the reins in driving.

There was decided loss of sensation over the left upper extremity, over the shaded area in the figure ; this was most marked, though nowhere quite complete, to touch ; loss of sensation to pain was less complete, and the temperature-sense and that of position were not affected. There was similar loss of sensation to touch and to pain over the left half of the face, the left conjunctiva and the corresponding side of the tongue and mouth. Over the rest of the body and limbs there was on the left side, limited by the middle line, a partial degree of anæsthesia less marked than in the left side of the face and upper extremity. Taste (quinine and constant current) was lost on the left side of the tongue, hearing and smell were very deficient in the left ear and nostril respectively, and there was considerable contraction of the visual field on the same side. The fundus and optic discs were normal, the pupils equal, reacting well to light and accommodation. The left arm was extended as far as possible and placed in a splint for the first day or two. He then attended for electrical treatment, and the arm was well rubbed with a stimulating treatment Tonics were given internally, and in a short time he recovered completely.

The clinical features of these cases leave no room for doubt that the patients were suffering from hysterical affections. In the first case the nature of the paralysis of the arm and hand, if considered apart from the other symptoms present, might have been doubtful, but its appearance was preceded for some time by slight attacks of vertigo, the description of which seemed to correspond with that of the milder kind of hysterical seizure, whilst no organic lesion of the peripheral nerves, brain or cord could explain the peculiar distribution of the anæsthesia, limited above, as it was, by a straight line encircling the arm, and leaving the index finger entirely exempt ; all forms of sensation being affected. In the second case the diagnosis was equally clear. The fits were those of the severer form met with in hysteria, and in addition to hemianæsthesia there was a well-marked hysterogenic zone, irritation of which produced an attack, slighter indeed, but presenting essentially the same features as those for which he sought advice. I had not previously come across so well marked an instance of the hysterogenic zone.

Common to all three cases was the unilateral affection of the organs of special sense on the same side as the anæsthetic areas, and in two of them there was hemi-anæsthesia. As other observers have pointed out, although the patients themselves sometimes complain of numbness and loss of feeling over one side of the body they do not always do so; the affections of sensation and of the special senses require to be looked for, and when found give valuable aid in diagnosis, for if ordinary precaution is taken in the investigation they furnish physical signs of the neurosis which are not likely to be vitiated by incorrect statements, as the patient is unaware of their import.

In the first two cases no history of any physical injury or severe mental shock could be obtained after careful inquiry, and they must therefore be regarded as instances of hysteria occurring apart from the action of any traumatic or other of the usual exciting causes. But when we search for the predisposing conditions, we find in the first patient the results of an infantile paralysis, which may be taken as indicating a neuropathic taint in his constitution; he was a drinker, and there was evidence of a lowered state of his general health in the tremor of the limbs, possibly of alcoholic origin, while he was moreover decidedly anæmic. The second man was of an excitable temperament, much given to indulge in highly emotional forms of religious excitement, so that in both these patients we may fairly conclude that they were in a condition of health which rendered them suitable subjects for the development of a neurosis. In the second patient this took the form of hysterical seizures and hemianæsthesia without any local paralytic or spasmodic affection.

In Case III. we have the more usual history of an injury as the exciting cause of an hysterical contracture, for this distinctly developed after the fall from his cab. But from the account which he gave us his health had been failing during the preceding twelve months, and he had been intemperate, so that in his case also the fall seemed to have merely brought into full action the already established tendencies to the neurosis.

Two of the cases have a feature in common, namely that the nature of their employment may have induced excessive fatigue in the limb affected, in the one case with paralysis, in the other with spasm. In each patient the limb most used in his work was the one affected, and in the cabman the arm was fixed by spasm exactly in the position in which he held the reins in driving The muscles involved were consequently those on which the greatest strain fell, which would continually be called upon to sustain greater efforts than the other muscles of the body, and the fatigue thus especially engendered in them may be supposed to have determined—in the general weakened state of the patients—the situation of the local affection. Why this should take in the one case the form of paralysis, and in the other, of spasm, it is difficult to see, but the explanation may lie in the different way in which the limbs were respectively employed, the mattress-maker in the operation of cutting out, constantly bringing into action the small muscles of the hand and forearm, which are easily tired, whilst the cabman maintained his arm for the greater part of the time in one position. The sense of fatigue arising from the constant fine movements suggesting in the former inability to continue the movement or paralysis of it, and in the latter the fixed position of the limb causing, at first cramp, and then a sense of inability to move the limb out of its strained position. It seems to me that, allowing for the influence of the shock of the fall in the last patient, that in all three cases the one common and important factor is an impaired nutrition of the cortical sensory and motor centres arising from a perversion of the general health. And that this impaired nutrition was an acquired condition, and not due to an hereditarily feeble nervous organisation seems to be clearly shown in two of the patients by the good family history which each of them gave; in the other the former occurrence of infantile paralysis and the statement that his mother was nervous render this doubtful. One may add that the mental state of these patients in no way resembled that which is perhaps still too commonly held to be characteristic of hysteria.

I have brought forward these cases as they are good instances of hysteria in working men, in whom the frequency of the occurrence of hysteria was not recognized till recent years. That it does occur fairly frequently in men of the working classes in all countries is now no longer doubtful. At the same time it is not so very common to find it fully developed in this country, even amongst a large number of hospital out-patients. If we may judge from the cases published in the journals, instances of hysteria in men are relatively more seldom met with in country districts and in the upper classes. Most probably this arises from the conditions under which a large majority of the poorer classes in our large towns live. The severity of the competition in the struggle for existence is greater than in the country; they live under bad hygienic conditions from absence of pure air and sunlight, and from overcrowding in their homes owing to high rentals, and, in comparison with the upper classes, are harrassed by many pressing anxieties from which the well-to-do are exempt. All such conditions are well adapted to produce that debilitated state of the nervous system on which the neuroses are grafted.

With regard to the question of the relative frequency of hysteria in men and women, it appears to me that although the number of cases of all kinds is very much greater in the latter, if we divide, for clinical purposes, cases of hysteria into two groups—the first comprising those patients in whom hemianæsthesia, with corresponding unilateral affection of the special senses is present as a more or less permanent feature with the addition of hysterical seizures, muscular spasms and paralyses, and the second group those milder cases in which paralysis, spasm, or any slight seizures exist without sensory affection—then we shall find that the proportion of the first group to the second will be relatively greater in men than in women, although the total number of cases is much less. In other words, when hysteria does appear in men it is more apt to assume a severe form.

Critical Digest.

ON HYPNOTISM.

BY C. LLOYD TUCKEY, M.D.

If the importance of a subject is to be measured by the amount of literature devoted to it we must conclude that the present revival of hypnotism is destined to mark an epoch in medical history, for to the number of books and articles in reference to it there seems to be no end.

The student will perhaps be relieved to learn that there is little that is new or original in the later works, and that the ideas and arguments permeating them are very similar in their tone, whether they emanate from German, French, American or English sources. The subject-matter is found to fall under the heads of theory, experiment and clinical experience, and the doctrines set forth show a leaning towards the teaching of one of two schools—that of Paris or that of Nancy.

Those writers who draw their inspiration from Professor Charcot agree in regarding hypnotism as an induced neurosis closely allied to hysteria, and therefore pathological; while those who range themselves on the side of Professor Bernheim refuse to see in hypnotism anything but a physiological condition almost identical in its nature to natural sleep. Charcot considers hypnosis a distinct entity marked by the presence of certain somatic phenomena; while Bernheim denies that there are any but psychical phenomena characteristic of the condition, and refers everything occurring during it to suggestion.

It is satisfactory to see that the fierce controversy which has been waged between the partisans of the two schools for the last seven or eight years shows signs of abatement, and it will probably ere long be terminated by mutual concessions on both sides.

As it is quite impossible to give in the limits of a short article a lucid account of all the recent books on hypnotism, it is proposed to apply ourselves in the following pages more especially to the consideration of two or three representative books which have recently appeared from the pens of two distinguished French writers.

Professor Bernheim's latest work[1] follows somewhat similar lines as his previous book,[2] and is in fact, as he says, a continuation of it. It is divided into two parts, in the first of which he devotes himself to the consideration of the theory of hypnotism and the *rationale* of cures of psychical origin generally, and in the second he deals with the practical application of his theories as demonstrated by the detailed report of over a hundred clinical cases.

Professor Pitres'[3] book is in two volumes. The first is given up to the discussion of hysteria, and is one of the most important works which has appeared on the subject within recent years; and the second is chiefly occupied with hypnotism and its phenomena. It is with the latter volume that we are at present concerned. Its title, and the fact that the preface is written by Professor Charcot, afford evidence of Dr. Pitres' mental bias.

Bernheim defines hypnotism as "The production of a psychical state in which the readiness of the mind to receive, and its ability to carry out, suggestion are greatly increased;" p. 15[2] and suggestion as "The act by which an idea is introduced into the brain and accepted by it as true," p. 24[1].

It may be observed that these definitions are very extended in their application, and that they do not mention drowsiness or sleep as necessary factors. Dr. Babinski[4] objects to them because they can be made to include many mental states in which, to his mind, there is no question of hypnotic influence; and this at once brings us to the point that much of the difference of opinion between the schools of Paris and Nancy depends upon failure on the part of their respective adherents to understand the position of their opponents.

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[1] *Hypnotisme, Suggestion, Psycho-Therapie*, Etudes Nouvelles, Paris, 1891, 1 vol. 8vo, p. 518.

[2] *Suggestive Therapeutics*, translated by Dr. C. A. Herter, Edinburgh and London, 1890, 1 vol. Oct. p. 420.

[3] *Leçons Cliniques sur l'Hystérie, et l'Hypnotisme*, Paris, 1891, 2 vols, 8vo.

[4] *Hypnotisme et Hystérie*, 8vo, Paris, 1891, p. 43.

Bernheim purposely leaves the definitions vague and comprehensive, because he wishes them to embrace a large number of instances in which the objective signs of hypnosis are entirely wanting. His contention is that hypnotism merely acts as the psychical preparation or vehicle for suggestion ; and he only induces it because he finds that in the hypnotic state the subject's mind can be concentrated on the idea of cure, and can be made to exert an influence over functions which, under ordinary circumstances, are beyond the control of the will. That the production of drowsiness is not necessary to cure of psychical origin is, he thinks, proved by the number of authenticated instances in which functional diseases have been removed by pilgrimages to shrines, and by faith healing, and he endeavours to propound a comprehensive theory which will cover all these cases.

When an idea is received by the subject as true, it tends, says Bernheim, to be realised in action, p. 31[1]. The thought of cold makes one shiver, and the remembrance of a disagreeable taste or smell produces a feeling of nausea ; the idea of dancing occasions a rhythmical movement of the feet, and the idea of pain evokes a feeling of local uneasiness. The vividness of the mental image and the extent of its corresponding accompaniment, whether motor or sensory, depend on the subject's receptivity and the strength of his imagination. Hypnotism increases mental receptivity and heightens the effects of ideo-motivity. The person in a normal state, if told that it is cold, will compare his present sensations with his previous experiences, and will accept or reject the propostion as his judgment dictates, but if profoundly hypnotised he will receive the statement as true, and will at once exhibit all the subjective signs of chill. If he be very receptive and sensitive, and the suggestions are forcibly repeated, he may even develop the *cutis anserina*, and other objective signs of extreme cold.

Neither Bernheim nor Pitres in the two books especially before us enter much on the discussion of the physiological basis of hypnotism and suggestion, but Professor Tarchanoff[5] has recently contributed a very clear and satisfactory monograph on the subject, and as the theory he propounds is that now generally held it may find proper mention here. Tarchanoff has continued the researches of Heidenhain[6] on the connection between hypnotism and cortical inhibition, and has come to similar conclusions on the subject. Briefly stated his theory is this. Under ordinary circumstances attention is being given and nervous energy is being expended in many directions in consequence of the arrival at the highest centres of impressions derived from all the avenues of sense, and a distribution and equilibrium of nervous action result. The person who is hypnotised by the method of fixation has his whole attention directed on a single sensory impression derived from one sense—the image of a brilliant object held before the eyes. This one-sided and excessive stimulation of one centre leads to the gradual inhibition of the cortical areas of the other senses, for it is impossible for more than one centre to be in excessive activity at the same time. As the subject's attention becomes more and more concentrated on the object he becomes less and less occupied with the life of relation until at length he is conscious of nothing but the visual impression. Over-stimulation of the visual centres leads to their exhaustion and consequent cessation of function, and a state of blank unconsciousness and senselessness ensues in consequence of impressions no longer reaching the cortical centres. The resulting condition is compared by Tarchanoff to what happens when all light is excluded from a room. If into this darkened chamber a ray of light is thrown it will shine with exaggerated force and brilliancy owing to the absence of other rays; and an idea suggested to the mind of a hypnotised subject will in a similar way assume abnormal weight and importance from the absence of contending or controlling ideas. It will become an impulse and tend to be realised with almost irresistible force.

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[5] *Hypnotisme, Suggestion, et Lecture des Pensées* (French translation), Paris, 1891.

[6] *Animal Magnetism* (English translation), London, 1888.

The state of mental vacuity in which the subject lies is well shown by one of Pitres' experiments, which also demonstrates how some persons respond to other than verbal suggestions. He hypnotised a young woman named Albertine, and she at once passed into a condition of profound trance in which she lay motionless and expressionless. On asking what she was thinking of she replied "Nothing," and her face showed no sign of intelligence. She was then placed in a kneeling posture with her hands clasped, and immediately her face, previously so dull and apathetic, lighted up and assumed an aspect of devotion resembling that seen in religious ecstacy, while her lips were seen to move. "What are you doing?" questioned Dr. Pitres. "Saying my prayers," answered the girl. When asked what she saw she replied "The Virgin," and proceeded to described her dress and appearance. In this case the attitude suggested the idea, and the thought provoked the hallucination, p. 155[3].

It is not difficult to understand how a suggestion thus become a dominant idea will affect the subject's conduct, at least for a time, and until it has been corrected or displaced by other impressions, but it is not so easy to explain the action of suggestion on organic processes and functions which are ordinarily beyond the influence of psychical agencies. It has, however, been abundantly shown by the researches of Beaunis,[7] Preyer,[8] Tamburini,[9] Krafft-Ebing,[10] and many other investigators, that hypnotic suggestion can be made to profoundly affect these. They have provoked local hæmorrhages in the form of stigmata from the skin, and discharges of blood from mucous surfaces. They have produced blisters from postage stamps by suggestion, and have rendered real blistering fluid inert by the same means. These results can of course be only rarely seen, as subjects of such susceptibility are not often met with, but Bernheim contends that if deep-seated and intense action is possible in exceptional instances, a sufficiently powerful, though less profound, effect is obtainable in many cases, and that hypnotic suggestion may thus be made to exert a curative influence in disease. He maintains that though he cannot evoke local hæmorrhage or blisters in an ordinary subject, he is able to exert such an influence over the vaso-motor and trophic centres that he can modify the blood supply and affect nutrition to a considerable extent. He therefore employs hypnotism not only in hysterical neuroses but in general constitutional and organic diseases ; and it is on this point that we find the greatest difference of opinion between Paris and Nancy. Charcot and his pupils admit that it is legitimate to induce a state they regard a pathological in certain obstinate forms of hysteria, and grant that the effect in these cases is often palliative and sometimes curative, but they refuse to consider the treatment applicable except to a very limited extent, and look upon it as a dangerous and uncertain remedy at the best. As for the employment of the minor grades of hypnosis—*le petit hypnotisme,* as they term it—they seem to regard it as entirely ineffectual and unscientific. And yet the great majority of people are only susceptible to the minor hypnotism, and the physicians who have taken up the question declare that they often cure their patients when the only symptom of hypnotic influence is a slight degree of drowsiness or torpor. Pitres quotes from the writings of Wetterstrand, Van Eeden, and Van Renterghem, De Jong, Schrenk-Notzing, and Tuckey, besides those of Bernheim and other French physicians, and expresses surprise and doubt at the statistics they have published, and gives his own experience in a number of cases in which he has tried hypnotic treatment. This experience seems to have been most unfortunate, for in the majority of cases where he did no harm he failed to do good, and in the few instances where apparent benefit followed it turned out to be only temporary or delusive.

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[7] *Du Somnambulisme Provoqué*, Paris, 1886.

[8] *Der Hypnotismus*, Vienna, 1890.

[9] *Riv. Speriment. di Frenatria*, vol. xvi.

[10] *Eine Experimentelle Studie, &c.*, Stuttgart, 1889.

Bernheim's statistics tend to show that if we are to accept Charcot's dictum that only hysterical persons are hypnotisable, and that the hypnotic state is practically a form of hysterical invasion, we must modify and greatly enlarge our conception of hysteria, for he finds that he is able to influence at least 90 per cent. of his hospital patients, and that about half of this number are affected to the extent of profound sleep or somnambulism. It is his experience, and also that of all the writers who adopt his views, that soldiers, sailors, agricultural labourers, and mechanics are the best subjects for hypnotism and the most amenable to hypnotic suggestion. Is it possible, he asks, that these persons are all hysterical or even neurotic? Are they not rather the classes who, from their occupations and family histories, form the least neurotic element of the community? Moreover nearly all children are hypnotisable, and hysteria is uncommon before the age of puberty. Many strong, robust and phlegmatic men fall at once into a state of complete unconsciousness with amnesia on waking and susceptibility to post-hypnotic suggestions, thus affording examples of the profoundest somnambulism, and yet they are not found to exhibit the somatic phenomena described by Charcot as characteristic of profound hypnosis. He therefore considers these cases as abortive, incomplete, and *fruste* forms of hypnotism.

Looking at the matter with an unprejudiced eye, it seems certain that Charcot's three stages are very rarely of spontaneous occurrence, though we need not accept Bernheim's view that they never occur except as the result of suggestion. Pitres was unable to show the students attending his lectures on hypnotism a single typical case, as he said there was not one in the hospital, but he affirms that he has seen several cases of spontaneous occurrence, and Tamburini, Lombroso and other Italian investigators assert that they have met with instances in which the possibility of suggestion was most carefully eliminated. The condition is therefore confessedly extremely rare, and is probably, as Tamburini says, an exaggeration produced by hypnotism of the increased reflex excitability characteristic of hysteria. Vizioli has recorded a case of *grande hystérie* in which Charcot's phenomena were elicited without hypnotism,

p. 128[3], and both Bernheim and Pitres quote cases where it was impossible to say which symptoms were due to hypnotism and which to hysteria. The fact that it is impossible to hypnotise a person during an access of hysteria is adduced by Babinski as corroborative of his assertion that the two conditions are identical, but it does not necessarily follow, for it is also impossible to hypnotise a person in the epileptic condition or in a state of intoxication. On the other hand Bernheim has frequently succeeded in influencing patients in hysterical paroxysms by suggestion and in guiding their fancies and movements, p. 175[1].

Bernheim does not consider that hysterical persons are the best subjects for hypnotism or the most satisfactory patients for suggestive treatment. Morbid auto-suggestion is, he finds, extremely apt in the first place to interfere with the acceptance of the suggestion, and in the second place to lead to a relapse. According to his definition a suggestion does not act unless it is accepted as true, and it is notorious that hysterical persons are often extremely unwilling to receive assurances of cure, and that if they receive a suggestion for a moment they often reject it on waking. Professor Forel states that in employing hypnotism our object is to bring the action of a healthy mind to bear on bodily disorder, and if the mind itself is diseased—as it so often is in hysteria—the very groundwork of success is wanting. The cures therefore effected in hysterical subjects are apt, as Pitres contends and Bernheim admits, to be of temporary duration. Unfortunately this is the case with all methods of treatment in such subjects, and it is not therefore surprising to learn that we have not found in hypnotism an infallible remedy for hysteria.

Nevertheless Bernheim, Dumontpallier, Déjerine, Babinski and other well-known hospital physicians report brilliant cures of hysterical neuroses, *e.g.*, megrim, pseudo-paraplegia, contractures, anæsthesia by hypnotic suggestion, and testify that the results have sometimes been lasting. Both Bernheim and Pitres point out that "suggestibility" is a variable quality, and that whereas some persons are open to suggestion without hypnotism, others refuse to entertain suggestions even when in a state of profound somnambulism. Bernheim, for example, quotes the case of a female patient who was under his care in the hospital for phthisis pulmonalis. He had not hypnotised her when one morning he stopped at her bedside and told her how she and the other patients had been disturbed the previous night by the misconduct of her neighbour, who had danced about the ward and made such a turmoil that it had become necessary to call the resident superintendent, who had come down in his dressing-gown and had with some difficulty induced the riotous patient to go back to bed. On making his rounds a few hours later he asked the woman how she had slept the previous night, and she at once replied that she and the other patients had been kept awake by the misconduct of her neighbour, who had made a great disturbance and had required the presence of the superintendent to quiet her. She then described the circumstances very much more fully than he had, and expressed herself as quite positive of the truth of her statements. Bernheim infers that from the facility with which he is often able to make persons, especially patients suffering from lingering diseases, believe absolutely groundless stories by simply repeating them in their hearing, that a very large amount of false evidence is manufactured by suggestions in this way, p. 175[2]. On the other hand, Pitres mentions how one of his patients, a woman, who was amnesic on waking and anæsthetic during hypnosis would yet refuse to perform any action to which she objected, p. 165[3], or to receive the suggestion of any hallucination or delusion.

Bernheim contends that hypnotism enables the physician to relieve symptoms and reduce them to their anatomical expression even in cases of incurable organic disease, and in replying to Dr. Gilles de la Tourette, who had twitted him with treating phthisis pulmonalis by hypnotism, and asked him whether he hoped to destroy the bacillus by suggestion, said—" I sometimes hypnotise patients suffering from tuberculosis not in order to suggest the disappearance of the tubercle, but to suggest that the patient shall sleep well at night instead of lying awake; and I find suggestions often succeed in restoring his appetite as well as in calming his cough, easing his pains, and improving his spirits. In doing this, it seems to me, that I fulfil the indications of the case, and if I do not cure him, I at least relieve his suffering. Sometimes, indeed, by modifying the conditions I increase his vital powers, enable him to resist the action of the microbes, and thus retard, if I do not cure, the disease," p. 209[1].

Bernheim points out that in many diseases of the brain and spinal cord, the symptoms have their origin not only in the area affected by gross lesion, but depend also on sympathetic derangement of neighbouring structures; and Pitres shows how the hemianæsthesia which sometimes occurs in cases of lesion of the posterior third of the internal capsule, does not depend upon section of the nervous fibres of the capsular region, but on simple functional derangement of adjacent ganglia, and in many respects resembles hysterical hemianæsthesia.

Babinski, in criticising the details of one of Bernheim's cases, also shows that the greater number of the symptoms accompanying a case of advanced cerebral disease may depend upon sympathetic disturbance, p. 41[4]. The case was one of left hemiplegia, with hemianæsthesia dating from an attack of coma a year before. The patient had post-hemiplegic lateral tremor simulating insular sclerosis, contracture of the left arm and leg, and exaggerated reflexes. He suffered much from dyspnœa dependent on emphysema. Hypnotic suggestion caused the rapid cessation of the tremor, relaxation of the contracture, return of common and painful sensation to the left side, return of the power to stand and walk. In time he became able to walk without a stick and with very little dragging of the limb. The improvement in the dyspnœa was very marked. The amelioration of all the symptoms continued for two years, during which time the man was kept in the hospital and occasionally hypnotised. The emphysema, however, gradually developed, cyanosis set in, and the patient died from pulmonary congestion and cardiac failure. The autopsy revealed foci of yellow softening in both hemispheres, upon the convexity of the left occipital lobe, and in the right hemisphere immediately behind the posterior extremity of the optic thalamus. Almost all the posterior portion of the external segment of the corpus striatum was destroyed, and its place occupied by a cavity filled with fluid. The medulla, pons medulla, and cord were not implicated, nor was there any degeneration of the pyramidal tract. Bernheim, in commenting on this case, suggests the possibility of suggestion having re-established the conductivity of sensory impressions by other tracts in place of those destroyed, p. 225[2]; but Babinski considers that hypnotic suggestion simply affected the hysterical element of the case. Whatever may be the correct explanation, the fact remains that a man suffering from grave organic disease of the brain was very greatly benefited by suggestion and enabled to walk long distances and feed himself for two years, after having been unable to for more than a year.

Several cases have been reported in English and foreign medical journals where the symptoms of locomotor ataxy have been greatly modified by suggestion, especially in respect to pain and co-ordination of movement; and Bernheim attributes the success of treatment by suspension to suggestion and the action of psychical causes. He has made a number of experiments, and has found that his patients were equally relieved whether the tension was real or only apparent, p. 61[1].

To explain the action of hypnotic suggestion on functions which are ordinarily beyond the influence of the mind, Bernheim evokes the theory developed by Dr. Hughlings Jackson and other physiologists, that the highest cortical centres not only subserve consciousness, volition and other faculties of higher cerebration, but also exert a controlling influence over the functions of the vegetative system. This influence, however, is not exercised under ordinary circumstances, but it is sometimes called into action in emotional states, and can be evoked by suggestion during hypnosis. Professor Delbœuf of Liége, imagines that the mind being withdrawn from the life of relation with which it is normally occupied, can be made to exercise during hypnosis a controlling influence over functions with which in the waking state it does not concern itself, and Liébeault, the venerable pioneer of hypnotic suggestion, supposes that in the hypnotic state we can direct nerve energy which is being discharged in wrong directions into the channels most beneficial to the organism. According to Bernheim every part of the organism is represented in the brain, and every gland and muscular bundle is ultimately innervated by a motor or other cell in the cerebral cortex, which is its *primum movens*. It is by influencing those cells that we are able through hypnotism to affect trophic function.

Whatever may be the true explanation of its *rationale* there seems no doubt but that in hypnotism we possess a remedial agent of great power and considerable usefulness. While admitting its power for good, some persons who have given attention to the matter have come to the conclusion that the risks attending the popularisation of hypnotism outweigh the good effects which are likely to follow its widespread employment, and their position is worthy of consideration.

If hypnotism is permissible at all, it is clear that it should be kept as much as possible in the hands of experienced medical men, and be used by them with caution and judgment. Its employment by ignorant or unscrupulous persons is certain to lead to similar abuses as brought mesmerism into disrepute.

It is refreshing to find that the schools of Paris and Nancy, while differing upon nearly every other point, agree about the necessity of regulating the use of hypnotism. In the interests of public health and morality it is to be hoped that England will soon follow the example of other countries, and that the public exhibitions of hypnotism will be forbidden by law.

Pitres fears that even the medical use of hypnotism is likely to do harm in many cases, and records instances in which the disappearance of one disorder has been followed by the substitution of another. For instance, he hypnotised a young woman for hysterical vomiting, and the suggestions cured it, but in its place appeared a severe headache, and when that was removed by renewed hypnotic suggestion the vomiting returned. This alternation of neuroses continued in spite of all treatment for months. Pitres also fears that repeated hypnotisation may, by unduly increasing the subject's mental receptivity, render him too suggestible, and credulous for the ordinary transactions of life. There does not, however, seem any ground for this fear if due care is exercised ; and the fact that Liébeault has practised hypnotism on a very large scale in a comparatively small town like Nancy for thirty years without causing any accidents, is in itself a strong argument in favour of the safety of the process. But the present writer is by no means prepared to acknowledge that hypnotism is such an absolutely safe procedure as it is assumed to be in some quarters, and he agrees with Bernheim and Pitres that it should never be employed except with the full consent of the patient and his friends, in the presence of witnesses, and with every precaution. It should not be induced more often than is absolutely necessary, and only with a definite therapeutic object in view.

Liébeault, whose testimony in matters of fact is unimpeachable, gives an experience which shows that hypnotic suggestion may originate as well as cure a troublesome complaint[11]. He relates that a child was brought to him suffering from a slight malady which he succeeded in removing by hypnotic suggestion. For some reason he suggested more frequent urination, with the result that in a few days the parents returned in a very disturbed state of mind, saying that a new complaint had developed worse than the former one, and that the child had wetted her bed each night for three nights. Dr. Liébeault hypnotised her again, and suggested the disappearance of the trouble, and the suggestion was efficacious.

The present writer has met with an instance showing that hypnotic suggestion may be distinctly dangerous if wrongfully applied. The patient was a middle-aged, very stout woman, with a weak and dilated heart, and subject to asthma. In addition to these chronic troubles she developed acute bronchitis, and a medical man was called in to attend her. It happened that he had been using hypnotism with success in his practice, and when he saw the patient struggling for breath he thought to relieve her by hypnotic suggestion. She was easily hypnotised and fell into a profound sleep, during which he suggested relief of the dyspnœa and quiet breathing. Under continued suggestions the respiration gradually became more shallow and quiet, until at last it ceased altogether. Recourse was had to artificial respiration, and in a few minutes the breathing had resumed its former character.

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[11] *Thérapeutique Suggestive*, p. 298.

Heidenhain thinks that even a healthy person might be placed in jeopardy if continued suggestions tending to interfere with respiration were made during profound hypnosis (p. 24[6]), and it is easy to understand how the risk is intensified when the breathing is already embarrassed by disease.

Pitres gives a very clear and interesting description of the phenomena of unilateral hypnosis, transfer, metallotherapy, and magneto-therapy which he has observed in the wards of the Bordeaux hospital.

For example, he describes the case of Paule C., a milliner, aged 20, of markedly hysterical temperament, and with complete right hemi-anæsthesia. She is easily hypnotisable by fixation of the eyes, and it is found that if only one eye is fixed she becomes hypnotised only on that side. When the left eye is fixed her left side becomes cataleptic whilst the right remains in the normal condition. If spoken to on the right side she replies in her natural voice that she is awake, and suggestions do not excite hallucinations; whereas, if addressed on her left side she answers in the typical hypnotic voice that she is asleep, and she can be influenced by suggestion. If told that she is in a garden and may pick the flowers, she will, if the suggestion is made in the right ear, look astonished and vexed, and say she is being made game of; but if the same suggestion is whispered into the left ear she will at once stoop and make the gestures of a person gathering flowers. She is not susceptible in her normal state to the action of metals placed in contact with the skin, but when hypnotised she is intensely affected by them. If, while in a state of unilateral hypnosis, a gold coin be placed against the normal side, no effect is produced, but directly it is brought in contact with the hypnotised side, she exclaims that it is burning her. It is the same in the muscular sphere. If the normal arm be raised it at once falls, whereas the hypnotised one remains in a state of cataleptic rigidity. In her normal state the right side is anæsthetic, but if a magnet be applied to the right forearm it is found that there is almost immediate transference of the condition to the left side, and the right side becomes sensitive. The transference once affected continues until the patient is aroused. This patient, who is one of a number of similar examples, possesses several hypnogenic zones all situated on the left side of the body, and pressure on any one of these at once induces hypnosis. Contact with the magnet causes transference of these zones to corresponding areas on the right side, and the same applies to the waking or hypno-frenatic zones.

According to Babinski and Luys, transference of symptoms may take place not only between the two sides of the patient but also between one subject and another. For example, Babinski found that when he placed two hysterical patients close together, the one affected with left and the other with right hemi-anæsthesia, and passed a magnet over the leg or arm, one of the subjects would become completely anæsthetic whilst the other would completely recover sensation.[12] Similarly with hysterical contractures, aphonia, and paralyses, they can often be transferred from one patient to another by the action of the magnet. Luys has carried this theory of transference to extreme limits, and applies it to subjects who are not hysterical and to conditions which are not merely functional. In the monthly periodical he edits,[13] one reads of cures of paralysis agitans and other intractable diseases by transfer, but his results lack confirmation in other quarters, and Pitres states he has been unable even to reproduce Babinski's experiments. Bernheim attributes all the phenomena of hypnogenic zones, transfer, and metallo-therapy to suggestion, and states that he has tried in vain to obtain them spontaneously at Nancy. Pitres, however, strenuously maintains that he has always most carefully eliminated the possibility of suggestion in his experiments, and his position as *doyen* of the faculty at Bordeaux, and the careful manner in which his book is written make one feel that it would be presumptuous to doubt the reality of the facts which he adduces, though they are sufficiently startling. It is often asked how it is that these things occur in the hospitals of France, whereas we never see them in those of England. Pitres' answer would undoubtedly be that it is because we do not look for them. Among the hospital patients of our large towns there exists much the same clinical material as one finds in Paris and Bordeaux. The numerous subjects on whom Pitres has experimented and obtained these phenomena are all highly hysterical women with ill-regulated minds and ill-nourished bodies. They are generally born of neurotic parents, and suffer from such hysterical symptoms as aphonia, anæsthesia, reduced field of vision, &c.

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[12] Soc. de psych. physiol., 1886. Soc. de biologie, 1886.

[13] *Revue d'Hypnologie.*

Berillon supposes that the phenomena obtainable during hypnosis supply an important key to the localisation of functions in the cortex. He describes cases in which pressure over the left parietal region produced contraction of the muscles on the right side of the body with motor aphasia, whereas pressure over the right parietal region caused contraction of the limbs on the left side without aphasia.[1] Pitres, however, refuses to accept this theory, and demonstrates that compression of other parts, *e.g.*, over the hyoid bone, is followed by similar results, and he attributes them to reflex muscular disturbance. In addition to hypnogenic zones, Pitres finds in a number of subjects on whom he has experimented other zones of a still more curious character. These he terms ideo-genic zones. He describes (p. 311, 3) the case of Eleanore F. She is easily hypnotised by fixation of the eyes, and when hypnotised exhibits several of these zones. Friction of the alæ nasi produces profound sadness;

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[1] "Hypnotisme Experimental, la Dualité Cérébrale et l'Indépendence fonctionnelle des deux Hémisphères Cérébraux." Paris, 1884.

she weeps, and exclaims, "I am in great sorrow. My poor mother is very unhappy." Friction over the mastoid process provokes an access of bad temper in which she stamps her feet and cries: "I am sure my mother won't let me go to Pauillac to-morrow." Friction over the temporal region is immediately followed by an outburst of laughter, in which she says: "No, it is too funny! This book is excessively amusing." Friction of the external occipital protuberance causes the patient to assume a threatening expression, and cry, "If I had a revolver I would kill him. The villain!" All these sentiments were apparently revivals of those she had really experienced. She had some time before been greatly chagrined by her mother's refusal to let her go to Pauillac. She had recently been reading an amusing book. She felt indignant with her father because he had abandoned her mother. But these zones were found to exist in other patients, and in all parts of the body. For instance, Théophile S. is a hysterical woman of twenty, and is also easily and profoundly influenced by hypnotism. In a state of catalepsy she exhibits many ideogenic zones. For instance, pressure or friction over the right clavicular region causes her to exclaim, "How hot it is here!" and at the same time she suits the action to the word by unbuttoning her jacket. Friction over the left submammary region induces a wish to sew, and she calls for her handkerchiefs that she may hem them. Friction over the right inguinal region makes her wish to curl her hair; while, if rubbed over the corresponding region of the left side she expresses a wish to write, and calls for pen, ink, and paper.

Friction over the anterior part of the left leg makes her express a wish to read, while over the right arm it causes her to see a bouquet, and to exclaim: "What a beautiful bouquet! please give it to me." And so on through over a score of regions, a friction over each one of which produces a corresponding mental state.

Pitres says that when he first experimented on this girl he formed the impression that she was shamming in order to appear interesting, but when he found that the effects produced were always the same, that they could not be altered by suggestion, and that contact with a magnet caused the transference of the zones from one side of the body to the corresponding area of the other side, he was forced to acknowledge them as genuine phenomena (3, page 312).

Pitres states that these zones are, as a rule, only operative during hypnosis, but he adduces cases in which they were demonstrable during the waking state, this being especially the case with erotogenic zones. Pitres does not accept the idea that there is a connection between certain skin areas and parts of the cortex, but prefers to suppose that the zones are first developed by auto-suggestion, and that association of ideas is subsequently sufficient to permanently fix them.

Summing up the cases, he writes that studying a phenomenon to-day is the best means of arriving at a conclusion to-morrow, and he is chary of giving explanations of the phenomena he has observed. He tersely defines the three stages of Charcot's grand hypnotisme thus: Lethargy is characterised by neuro-muscular hyper-excitability with general relaxation and psychical inertia. Catalepsy is marked by cataleptic plasticity of the limbs with susceptibility to suggestion by attitude, and depends on spinal automatism. Somnambulism is dependent on cerebral automatism, and is characterised by cutaneo-muscular hyper-excitability.

Bernheim says but little, and Pitres nothing at all, on the subject of the treatment of drunkenness and dipsomania by hypnotic suggestion, but according to many other writers this is the field in which suggestion shows itself most powerful, and is the sphere in which its effect is most strikingly beneficial. Bernheim states that he has cured four or five cases of morphinomania which had resisted other treatment (1, page 228). Berillon finds hypnotic suggestion a valuable reformative agent in the case of vicious children. Auguste Voisin is almost the only observer who credits hypnotism with being useful in the treatment of insanity, and the Commission of Italian alienists and neurologists, who have been thoroughly investigating the subject, have reported that they find hypnotism inapplicable in cases of true insanity. At the same time they give it their opinion that in dipsomania. and in certain hysterical conditions simulating insanity it is often efficacious.[1]

It will be seen from the above notice that Professor Pitres does not advocate hypnotic treatment except in special cases, but he considers that the question should be studied by medical men, and he advises his students to master its theory and practice in order that they may know how and when to apply it (3, page 415).

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[1] Vide paper by Dr. G. Seppelli, translated from the Archivio Italiano in *American Journal of Insanity*, April, 1891.

Abstracts of British and Foreign Journals.

Thyssen on Astasia-Abasia (*Arch. de Neurologie, t.* xxi.).

THE combination of symptoms (syndrome) about to be considered is first alluded to in a work by Jaccoud,[1] where it is described as "Ataxia from want of automatic co-ordination." Lebreton[2] evidently alludes to the same condition in a thesis on Hysterical Paralysis, in these words: "It often happens that the patient, when in the recumbent posture, can execute extensive movements with the legs. But so soon as she is ordered to walk, after taking a few faltering steps the legs begin to fail, and eventually give way under the weight of the body; it would appear as though at a given moment the accumulated store of nervous energy had become exhausted." Some fifteen years later the symptom is described by Charcot and Richer[3] as a "special variety of motor feebleness of the legs from want of co-ordination in relation to position in rest and progression." Weir Mitchell[5] describes the same condition under the name Hysterical Motor Ataxia (1885.) Erlenmeyer,[6] Henoch,[7] and Romei[8] also allude to it.

It was, however, Paul Blocq[9] who, in 1888, made the first exhaustive study of the condition, and established its independence. From the consideration of cases which came under his own observation, and of previously recorded cases of similar type, he deduced a new pathological state (type morbide), for which he suggested the name Astasia-Abasia, which has been since more or less generally adopted.

In the same year Charcot[10] again drew attention to the condition, in a girl of 11 years, at the Salpêtrière, and for the first time used the term Astasia-Abasia in describing a case of this affection in a little boy.

More cases were observed and described by Souza-Leite,[11] Paul Berbez,[12] Grasset,[13] Berthet,[14] Salemi Pace,[15] Brunon,[16] and Charcot.[17] Further additions to the literature of the subject were made by Fr. Helfer[18] (a case of Astasia-Abasia following on Influenza); by Ladame,[19] (a case in which the affection was

paroxysmal). Professor Binswanger[20] has more recently contributed a remarkable paper entitled "Ueber psychisch bedingte Stœrungen des Stehen und des Gehen." Finally, we must mention Mœbius' contribution.[21]

The following is a brief account of the author's cases.

Case I. (Jan. 1888.)—A girl of 11 years; epileptic since three years, trouble in walking of same date. Walking became impossible in November, 1889, after an attack of typhoid fever, but recovered partially. When first seen standing and walking were accompanied by marked staggering (without giddiness). On the other hand, the child could always hop, skip, or go on all fours without any difficulty. Examination of the legs when sitting revealed nothing abnormal—no anæsthesia, no inco-ordination. It had been noticed on several previous occasions that after an epileptic seizure there was a decided narrowing of the field of vision, and a marked improvement in walking for a day or two. On February 22nd, a few hours after a seizure, walking was almost natural, the child only staggering, or rather swaying a little, when turning to the right or left. Closing the eyes did not aggravate the unsteadiness. She continued to hop with perfect ease. The patient was subsequently lost sight of.

Case II. (1888.)—A girl of 12½ years, of neuropathic stock. She was seized in 1887 with giddiness, vomiting and headache, accompanied by spinal hyperæsthesia of such severity as to suggest cerebro-spinal meningitis. In July, 1888, her mother noticed disordered movements of the legs when she attempted to make her stand. About the same time she developed convulsive seizures, preceded by a cephalic aura, with tendency to general contracture. In October following, the patient was seized with unconsciousness whenever the dorso-lumbar portion of the back of her couch was removed, or when it was attempted to make her stand up. It sufficed to sit the child up with the back supported, or to lay her flat on her back, for consciousness to return at once. When sitting up and conscious she is able to move her legs without inco-ordination whilst her eyes are open, but with closed eyes no movement of the legs is possible. During a stay at the seaside she has been able to bathe and swim, in the prone position, without losing consciousness. She spends the day in a wheeled chair with treadles, in which she moves about with ease and rapidity. She presents several hysterogenetic areas which are tender on pressure—ovarian, mammary, spinal, facial and vertical. As a result of treatment by douches and isolation her condition began to improve in 1889, and after six months of functional impotence the child began to walk again, at first with a tottering gait, but eventually without difficulty.

Case III.—Miss G. C., aged 19 years, came to the Salpêtrière on October 28th, 1888. She has a strong neurotic family history. She stated that she had for some time been liable to attacks of weakness (faiblesses) when crossed in any way. Since six months she has suffered from weakness of the legs, worse during the last month, and is unable to stand without support. Since one week choreic movements of the face and right arm have been noted. The lower limbs offer a normal resistance to passive movements of flexion and extension. Muscular power is somewhat diminished on the left side. She leans back in her armchair, and cannot sit up without supporting herself with both arms. She cannot sit up unaided, or rise from the sitting posture. She is unable to stand without support, and even then sways backwards and forwards, and from side to side. When standing she throws herself back on the heels, and the upright posture is further interfered with by inco-ordinate movements (mouvements contradictoires) and prevented by the right-sided paralytic chorea. In walking she drags the right foot (typical gait of hysterical hemiplegia). Subsequently the attacks ceased, and the patient was lost sight of.

Case IV.—Male, aged 26 years. After a four years' residence in Tonkin, where he contracted dysentery, he was admitted to hospital on his return in October, 1889, suffering from cramps in the lumbar region. His condition on November 6th was the following: lumbar pain, paresis, dyæsthesia of legs as low as knees; frequent cramps in legs; some incontinence of urine; exaggeration of knee-jerks; epileptoid trepidation.

At the same time walking was becoming affected, and when ordered to walk he would jump about as though executing a dance. In February of the following year walking had become quite impossible; as soon as his feet touch the ground he springs from toe to heel as if he were on springs. In March he had ceased to attempt to walk. In April there was a marked improvement, and in the following month standing and walking had become possible, though still very saltatory; in July the saltatory movements were daily growing less marked. Dr. Brissaud, who had previous charge of this patient, communicated the following particulars respecting him: "The patient, when he first came under M. Charcot's care, was no longer suffering from *Rythmical Saltatory Abasia*. But at an earlier period — in January—he had spasmodic seizures, which, however, were independent of abasia. When extended the patient would occasionally be seized with rhythmical tremor precisely similar to that which occurred during walking. This tremor, which came on spontaneously at times, and could be provoked by forcible extension of the leg or flexion of the foot, was closely analogous to epileptiform tremor. Sometimes a sudden removal of the bed-clothes sufficed to initiate the tremor in both legs."

In this respect therefore this patient resembled the subjects of saltatory spasm. He was astasic and abasic only because the weight of the body on the soles of the feet was necessary to cause the spasm. As a matter of fact his attitude when standing and during walking showed no evidence of inco-ordination, but was well under control and not interfered with in any way by the tremors. Gradually the saltatory spasm subsided, and at the present time there only remains some rhythmical abasia which will shortly disappear.

After recording eleven cases of the affection M. Blocq goes on to say: "The onset is rather sudden in the majority of cases. After a sudden emotion or slight traumatism the condition comes into existence at once, or more gradually within the space of twenty-four hours. Not rarely at this period it is preceded or accompanied by pains, such as cephalalgia or rachialgia."

In Charcot and Richer's observations (1883) we read as follows: "this condition develops suddenly after an emotion or traumatism." But in connection with a case he demonstrated last year at the Salpêtrière, Charcot observes: "the neurasthenic disorders sometimes develop suddenly in response to some determining cause, such as a mild attack of rheumatism in which the *physical* disturbance is far in excess of the *psychical* (*sic.*); or during convalescence from acute disease attended with marked bodily prostration, *e.g.*, enteric fever or protracted labour; or again, after intoxication by carbonic oxide as in the present instance."

In this last trepidation in walking came on twenty-two days after the intoxication and appears to have been determined by his jolting against a passer-by.

In Romei's case a child of 11 years developed astasia and abasia within twenty-four hours of a fright. Souza-Leite mentions "a small epidemic of Astasia-Abasia" in which three white women, a mulatto, and a negress were affected.

Prof. Henoch has seen it develop slowly in a child of 7 years, from onanism. The patient experienced difficulty in walking, with intermissions for the first eight months, afterwards permanently.

A lady of 27 years, under the care of B. Salemi Pace, was seized suddenly after a walk, the only discoverable predisposing cause being weak health of several years' duration. In Berthell's case—a girl of 23—the affection followed on hysterical attacks. Binswanger's patient—a business man of 55—was seized quite suddenly as he was sitting down to dinner. He recovered after a while, but subsequently the disease re-appeared in an intermittent form. In a second case (æt. 58) by the same observer, the onset was also quite sudden. In his third case the patient, a music master, aged 35, fell down in the street, and on being raised up was astasic and abasic. Overwork is mentioned as a predisposing cause. Ladame mentions the case of a circus rider who felt unwell during a performance, and four days later suddenly lost the power of walking for a few minutes. For the next four years there was no further evidence of astasia-abasia, but for the last twenty years the condition has returned whenever the patient attempted to walk more than a hundred yards. In this case there was a history of privation, unusual fatigue and physical overwork. A girl of ten years observed by Helfer was siezed on attempting to rise from her bed after influenza; she was cured within five months.

In the author's cases the onset has usually been gradual. Of Blocq's eleven cases the onset was sudden in five.

From these data we may conclude that although the onset is sudden in some cases, or attains its full development in the course of twenty-four hours, it is also frequently slow in its evolution, resembling in this the onset of traumatic hysteria.

Persons of all ages appear to be liable to the affection; the majority of the cases belong to the earlier periods of life, but at least three have occurred in elderly persons.

In addition to the cases quoted or related above, in the course of last year five or six adults suffering from the condition have come under observation at the Salpêtrière. Sex and race do not appear to act as predisposing causes.

Hereditary taint is one of the most constant factors in etiology. Nervous diseases in the parents are of frequent occurrence, and rheumatism is also common. As a matter of fact, the majority of those attacked are predisposed to nervous affections, and this is well borne out by the large majority of the cases quoted above.

As regards symptomatology, all observers draw attention to the fact that the trouble concerns exclusively the combined movements involved in standing and walking, which may be interfered with in varying degree, whilst on the other hand, in the horizontal position, the patient retains full muscular power and complete control over his limbs.

This main fact of the integrity of power and control in the horizontal station, and the consequent non-appreciation of the combined muscular efforts required in standing and walking, justify the use of the term "astasia-abasia."

The several features of the condition are demonstrated by making the patients stand up and walk. In some standing and walking are absolutely impossible; as soon as the patient is put on his legs they give way under him, and he falls to the ground; but the same individual will get along perfectly on all-fours, or hopping, or will swim without difficulty. Such a person when supported on both sides will put forward his legs much in the manner of a child learning to walk (Blocq). In others standing and walking are both possible, but attended with great difficulty.

The subjects of this complaint devise all sorts of methods of progression. Some hop along like magpies or push with their feet the chair on which they are sitting. A remarkable case occurred at the Salpêtrière, in which the patient could execute a stage-walk (marche dramatique à très grands pas), but was unable to walk quietly. In many cases associated hysterical manifestations reveal the origin of the trouble, but its existence as a symptom of organic spinal disease is not impossible.

Professor Binswanger affects surprise at Blocq recognising three forms of the affection, according as the power of standing and walking is abolished, diminished or modified, and considers the number of divisions excessive in view of the small number of cases observed, for Binswanger only admits that three of Blocq's eleven cases come within the terms of Charcot's definition of the malady; the troubles observed in the other cases being attributed by him to anæsthesias and hyperæsthesias. Of this more anon. Grasset, taking count of the quality rather than of the degree of disturbance, describes three types, according as the trouble is due to weakness to inco-ordination or to rhythmical movements (rhythmical choreic type).

Charcot, in his lecture (1889), adopts the following division:—

$$\text{Abasia.}\begin{cases}\text{Paralytic}\\ \text{Ataxic}\qquad\begin{cases}\text{Tremulous,}\\ \text{Choreiform,}\end{cases}\end{cases}$$

according, as standing and walking are impossible, or ataxic, or attended by tremor or choreiform movements. This table roughly indicates the chief heads under which the doubtless numerous clinical varieties of this affection will naturally group themselves (Charcot). Ladame's case already introduces a complication, and necessitates the addition of a sub-division for paroxysmal astasia-abasia. Moreover, Brissaud's case does not come within any of the existing groups. The author suggests the following classification :

$$\text{Astasia-Abasia.}\left\{\begin{array}{l}\text{Paroxysmal}\\ \text{Continuous}\end{array}\right.\left\{\begin{array}{l}\text{Paraplegic}\\ \text{Ataxic}\end{array}\right.\left\{\begin{array}{l}\text{Tremulous or Trepidating,}\\ \text{Choreiform,}\\ \text{Saltatory,}\end{array}\right.$$

which may have to be further amplified.

In connection with Souza-Leite's observations, Binswanger maintains the necessity of submitting all recorded cases to a searching criticism, and points out the dangers which may arise from describing a disease in terms of its most striking symptom. The author admits that the two cases in question differ somewhat from the types above laid down, but thinks that Binswanger's objection is fairly met by these words of Charcot : "you must not expect to meet in this domain, or in any other indeed, with pure forms or perfect types."

To Blocq and Grasset, amongst others, is due the credit of having differentiated these symptoms of astasia-abasia from associated conditions, and of having demonstrated that these latter are not the cause of the troubles we are here considering.

It is well known that in many cases of perverted sensibility of the lower limbs the gait is in no wise affected. Moreover, it is not easy to see why sensory troubles, when present, should only affect the mechanisms of normal walking, leaving intact the mechanisms involved in other modes of progression. Sensory troubles when they co-exist are therefore to be considered as hysterical manifestations and not the real cause of the abasic phenomena. As regards differential diagnosis : the inco-ördination in tabes dorsalis and Friedreich's disease attends all movements of the lower limbs. The hysterical ataxia of Briquet and Lasèque occurs only when the eyes are closed. In hysterical paraplegias with complete muscular relaxation and impotence the paralysis is equally marked in the recumbent position. The muscular spasms of rhythmical chorea are characterised by their cadence and regularity. In the reflex saltatory convulsion of Bamberger, spasmodic paralysis, exaggerated reflexes and epileptoid trepidation are prominent symptoms.

Charcot and Blocq have put forward to explain the phenomenon an ingenious psychological hypothesis which has been opposed by Binswanger and criticised adversely by Mœbius. The pathogeny of the French authors is based on our knowledge of the way in which acquired movements become organised. The greater part of the centres for combined muscular movements such as standing, walking, running, swimming, the playing of various musical instruments, &c., are localised in the brain and spinal marrow. On their hypothesis the spinal centre contains the "materiel d'execution," and is set going by the corresponding cortical centre, by the "psychological memory," which determines the kind of impulse necessary to produce the required movement. For standing and walking, which are slow of acquisition, Blocq postulates the existence of differentiated cortical cellular groups which are put in relation by special commissural fibres with corresponding groups of cells in the spinal centres. These cortical groups initiate the stimulus in response to which the spinal centres act automatically. These hypotheses have received a certain amount of experimental confirmation, notably from the observation of automatic movements (flying, swimming, &c.), of certain animals after decapitation. Salemi Pace is of opinion that the cord plays a preponderant part in the causation, and in his own case came to the conclusion that there was a peculiar and partial modification of function in the spinal cord characterised by "dynamic and static motor amnesia." The author is inclined to adopt the views of Blocq, who develops Charcot's ideas, the hypothetical nature of which is fully recognised. The several mechanisms concerned in the performance of walking, standing, jumping, &c., probably contain two centres, or differentiated cellular groups, the one cortical, the other spinal, which are connected by commissural fibres. The spinal group, without doubt the more complex of the two, governs automatic unconscious execution of the co-ördinated movements necessary to the accomplishment of each function; whereas the cortical group has the relatively simple duty of the voluntary issuing of orders to initiate, accelerate, slacken, or arrest the activity of the spinal group. In other words, the *psychological* memory of the acts necessary to set going or to check the nervous apparatus are located in the cortical centre, whilst the *organic* memory which presides over and controls all the details of the prescribed movements has its seat in the spinal centre. Hence in each case the question arises whether the disturbing cause of normal function is to be sought in the encephalon or in the spinal cord.

It is commonly known that fright may cause paralysis of the legs; and fright is precisely one of the determining causes of astasia-abasia, which would thus be an emotional paralysis succeeded by an auto-suggestion of motor impotence. An inhibition of the cortical or spinal centres controlling standing and walking, would serve to explain the mechanism of such a process. Blocq comes to the conclusion that the change is purely dynamic, but still is localised in the nervous centres in such a manner as to give rise clinically to symptoms identical with those due to organic lesion of the same area. These very simple and ingenious hypotheses Blocq has been able to verify experimentally by suggestion.

After excluding the cases which present muscular contractions, trepidation and clonic contractions capable of disturbing the functions of standing and walking, together with those in which pain may have prevented movement, Binswanger places the few remaining cases in the large class of "Zwangvorstellungen," or "obsessions," or under the head Hypochondriasis.

The outcome of Binswanger's criticism of the theories of Charcot and Blocq is that he regards astasia-abasia as a state in which the disordered feelings of the organs maintain a hypochondriac preoccupation, or in which well defined causes compel attention to be fixed suddenly and exclusively on an imaginary motor or static impotence.

In order to explain the "obsession," (*hypochondrische vorstellungskreise*) with which these patients are seized, according to his view, this author quotes Spinoza; the manner of thought of these patients appears to be strongly influenced by their melancholic turn of mind which is in turn kept alive by the abnormal state of their viscera. He is unable to see what objections there are to classing "obsession" as a symptom of hysteria, since in both conditions bodily action is powerfully influenced by imagination and *vice versâ*.

The author maintains, however, that a line can be drawn between "emotive paralysis" or "paralysis dependent on idea" which occurs in persons of hysterical diathesis as a result of traumatism, preoccupation, or emotion, on the one hand, and "obsession" pure and simple on the other, which affects quite a different category of neuropathic individuals.

The only point in common between the two states is that in both the disturbance is of a psychopathic order. Nevertheless, without insisting on the considerable clinical divergence in the two conditions, attention may be drawn from a psychological stand-point, to the essential difference between the mental troubles in the two cases.

Whereas the hypochondriac is "attentive" even to the point of "obsession" the hysteric is essentially absent-minded. The one concentrates all his faculties on his disease, which is the reason why suggestion is powerless in his case; the other takes no heed of his complaint, and is for that reason generally accounted so favourable a subject for treatment by suggestion. Astasia-abasia is an unconscious, obsession a conscious process.

Mœbius clearly recognises the need of distinguishing between the "inability to will to stand" of the hysteric, and the fear of falling of the subject of "obsession." The "I cannot" of the former is in reality "I cannot will to." As showing the effect of "loss of power to will," when Blocq told a hypnotised subject "you have lost the power of walking," he caused a total loss of motor power; but when he said to him "you no longer know how to walk," the loss of power amounted only to inco-ördination. From another point of view a sort of experimental realisation of abasia lends support to Blocq's theory. It was noted by Steiner that after removal of one of the cerebral hemispheres of the dog-fish, the animal swims in a circle. If it is then allowed to pursue this abnormal (abasic) mode of progression for some time, its lower centres become organised in such a way that, after removal of the other hemisphere, the same animal still continues to swim in a circle.

Mœbius justly observes that the hysteric is conscious that he cannot walk without knowing the reason why, whereas the hypochondriac cannot understand why he is afraid to walk. Again agoraphobia may develop concurrently with neurasthenia, and the latter condition is frequently associated with hysteria, so that the two conditions may co-exist in the same person; it might happen, therefore, that after the sudden disappearance of astasia-abasia, agoraphobia which is always more slow to disappear, would persist alone.

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[1] Jaccoud.—*Paraplégie et ataxie du mouvement*, page 653, Paris, 1864. [2] Thèse de Paris, 1868. [3] Charcot et Richer.—*Sudi una formă d'impotenza motrice degli arti inferiori per diffetto de coordinazione (Medecina contemporanea*, 1883, p. 6, no. 1.) See also Charcot: [4] *Lezioni cliniche dello anno scholestico*, 83-84, relatte del dotr Millotti, Milano, 1885. [5] Weir Mitchell.—*Lect. of diseases of the nervous system especial in women.* (Philadelphia, 1885, p. 39). [6] Erlenmeyer.—*Ueber statische Reflexkrampf*, 1886, Leipzig. [7] Henoch.—*Hystérie des enfants*, Paris, 1885. (Translation.) [8] Dr. Serafino Romei.—*Paraplegia infantile nel solo atto della ambulatione. (Gazetta degli ospitali*, no. 76, p. 605.) [9] Paul Blocq.—*Sur une affection caractérisée par de l'astasie et*

de l'abasie, Paris, 1888. *Archives de Neurologie*, no. 43-44. [10] Charcot.—*Leçons du Mardi*, 24 janvier, 1888. [11] Souza Leite.—*Reflexions à propos de certaines maladies nerveuses observées dans la ville de Salvador : faits d'ataxie et d'abasie. Progrès médical*, XVI., Paris, 1888. [12] Paul Berbez. — Gazette *hebdomadaire*, 30 novembre, 1888. [13] J. Grasset.—*Leçons sur un cas d'hystéric mâle avec astasie-abasie.* Montpellier méd., mars, 1889. [14] Berthet.—*Sur un cas d'astasie et d'abasie.* (Lyon méd., juillet, 1889.) [15] Salemi Pace.—*Amnesia parriale spinale. Gazetta Sicula*, Anno IX., p. 182. [16] Brunon.—*Normandie médicale*, 1889. [17] Charcot.—*Leçons du Mardi à la Salpêtrière*, 1888-1889, 5 mars. [18] Fr. Helfer.—Jahrb. CCXXVI., p. 112. *Med. Gesellschafft zu Leipzig.* [19] Ladame.—*Arch. de Neurologie*, XIX., p. 40, 1890. [20] Prof. Dr. Otto Binswanger.—*Berl. klin Wochenscrift*, 1890, no. 21.—*Aus der psychiatrischen Klinik zu Jena.* [21] Mœbius.—*Ueber Astasie-abasie*, 1889.

W. PASTEUR, M.D.

Current Nerve Anatomy and Physiology. By ALEX HILL, M.D., Master of Downing College, Cambridge.[1]

PERIODICAL.

1. **Journal of Comparative Neurology.** (*Robert Clarke & Co., 63, W. Fourth Street, Cincinnati, U. S. A. Price* $3.00 *per annum*).

Since the last collection of abstracts was published in BRAIN, a new journal, devoted to Comparative Neurology, has made its appearance. It is to be published quarterly, and the Editor, whose work, as shown by the first two numbers, gives an excellent promise for the future, undertakes to supply his readers with lists of current neurological literature, resumés of important papers and synopses of those of less general interest. The titles of the papers which appear in the first two numbers are given below, but as they only reached us when this number of BRAIN was passing through the press it was impossible to abstract their contents.

ANATOMY.

2. **Certain Recent Researches into the Anatomy of the Central Nervous System.** WALDEYER (*Berliner klin. Wochenschr.*, July 13, 1891, p. 691).

It may be interesting if this short summary is translated in extenso.

"The investigations under discussion were opened by the record breaking work of Golgi, in Pavia, and carried further especially by S. Ramón y Cajál in Barcelona and by Kölliker. To these must be added the embryological observations of His, the comparative and histological investigations of G. Retzius in Stockholm, Fr. Nansen in Bergen and Biedernan. The following are the chief results :—

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[1] It is hoped that the retrospect for 1891 may be completed in the Spring Number. The abstracts now given are not necessarily prior to many which will appear in the complete retrospect.

1. "The axis cylinders of all nerve fibres are connected at some part of their course with nerve cells. These places can be described as their origins since, embryologically, the axis cylinders sprout out from the cell, and thence extend centrifugally as well centripetally (*His*).

2. "The ending of all axis-cylinder processes (nerve-processes), whether central or peripheral, appears to be by means of the so-called nerve-branches (*S. Ramón y Cajál* and *Kölliker*). These entirely resemble the antler-like (*W. Kühne*) end-branching of muscle-nerves. A certain number of authors assume that the twigs of neighbouring end-branches are immediately connected together in a network, and also that in addition to the cellular origins an anastomotic connection of axis-cylinders is present throughout the central organs (*Golgi* and his pupils, *Martinotti* and *Sala*, on comparative grounds, *Bela Haller, et al.*), while the greater number (*S. Ramón y Cajál, Kölliker, His, Nansen, Retzius*) can nowhere see an anastomotic connection, so that the different end-branches form within the central organs a thick felt work (nerve-felt, neuro-pilema, *His*), their ends, nevertheless, always being free. If this be true, the transfer of stimuli from nerve to nerve must be regarded as actio in distans."

[Attention has not hitherto been called to the nuclei, granules or bi-polar cells which are found upon the end-branches. They are notable features in muscle-plates, in the embryonic substantia Rolandi, in the cortex of the cerebellum and cerebrum. It may be that a long fibre which starts from a nerve cell ends after dividing by connection with "granules." It is also possible that the protoplasmic processes of nerve cells do not form a permanent network but have a certain mobility, forming variable connections along the lines traversed by nerve impulses (*Hill*).

3. "As *Golgi* discovered, the axis-cylinders also bear lateral branches—these have been traced everywhere, especially by *S. Ramón y Cajál* and *Kölliker*. *Ramón* termed these side branches collateral. Quite different, apparently, are those branches which are to be seen on the posterior roots, to which *Ramón* assigns the following relations :—Each posterior (sensory) root fibre on entering the spinal cord splits in a T shape, one branch running up and the other downwards longitudinally in the cord. Collateral branches originate in fairly regular manner from both these longitudinal branches, as well as from the entering root

fibre. They enter the grey substance and there branch and end freely. Such collateral branches spring also from the anterior root fibres, from the other longitudinal root fibres as well as from the nerve fibres of the medulla oblongata (*Kölliker*), the cerebrum, and cerebellum.

4. "Thus a nerve element, a nerve entity, or 'neuron,' as I propose to call it, consists as the results of observation show (if we do not allow the existence of a reticular connection) of the following pieces :—(*a*) a nerve cell, (*b*) the nerve process, (*c*) its collaterals, and (*d*) the end-branching. This simplest form of the neuron appears, in fact, to be exclusively present in the higher invertebrates as investigated by Nansen, G. Retzius, Biedermann.

5. "The neurons arrange themselves according to Golgi's investigations, which are confirmed by all other people, in two forms, or let us rather say sizes, the 'long' and the 'short.' The long ones send their nerve-process either outwards from the central organ to the periphery in the form of the axis-cylinder of motor, secretory or sensory nerve fibres, or as long tracts through the whole central nervous system for the purpose of tying together its distant parts. The short or local neurons, as we can call them, do not send their axis-cylinder processes farther than the grey substance in the neighbourhood of the cells which belong to them. Golgi has started the hypothesis that they have sensory functions, yet this is not at present certain. We must rather consider that we do not yet know their meaning.

6. "The cells of the neurons in higher animals have, in addition to the nerve-process, a greater or smaller number of other processes, which richly branch (Dendribes of *His* ; Protoplasmic Processes, *Deiters*). Their meaning is still uncertain. Golgi and his pupils, as well as Nansen, deny that they have a nervous function ; rather is their purpose for the nourishment of the cell. As a matter of fact, one often sees them branch for a long distance in the white substance, where they apply themselves to the supporting tissue and the blood vessels. Nansen is, as far as possible, in opposition to Golgi, and assigns to the nerve cells themselves merely a nutritive function. Ramón y Cajál, Kölliker, and the greater number of other investigators hold, on the other hand, that the cells have undoubtedly nervous functions, but deny with more or less assurance a similar meaning to the dendribes.

7. "Also for the neurogleia (*R. Virchow*) have His, Golgi, S. Ramón y Cajál, Kölliker, and M. v. Lenhossék brought many new facts to light. Two kinds of elements must undoubtedly be

recognised, the ependyma-cells and the gleia-cells (spider cells of Jastrowitz). Ramón and Lenhossék derive the latter from the former. These (the ependyma cells) reach in embryos and in lower vertebrata throughout life from the ventricular surface to the pia-surface. So found, for example, Oyarzun recently in the fore-brain of the frog, as recorded by Edinger in a work issued by the senckenbergischen Gesellschaft. All gleia-cells and also the ependyma cells branch but do not anastomose. Whether in addition to these elements, which have the same source of origin as the neurons (the ectoderm) genuine connective tissue elements appear in the central nervous system (as Götte, Duval, Stricker, and Unger and others think) is a question which is variously answered. His thinks that they soon separate themselves."

3. **Sections through the Human Central Nervous System.** Kronthal (*Berlin, Speyer and Peters*, 1890, 17 *Photogravure plates*).

Some of the sections are printed without enlargement, others are magnified. They are cut in various planes, and although stained by several different methods, they are all printed in dark blue-black.

4. **Hughes, Nerves of the Human Body.** (*Edinburgh*, 1890. *E. & S. Livingstone*, 10 plates, 4to).

Topography.

5. **Cranio-cerebral Topography.** Assaky (*Clinica Bucureski*, 1890).
6. **Cranio-cerebral Topography.** Debierre (*Gazette Hebdomadaire de Médicine et de Chirurgie*, xxviii. 14, 1891, pp. 159-161).

Comparative.

7. **Comparative Anatomy of the Human and Simian Brain.** Wilder (*Philos. Soc. of Washington*, April 14, 1890).
8. **The Central Nervous System of Rodents.** Herrick & Tight (*Bulletin of the Scientific Laboratories of Denison University*, vol. v. 19 plates).
9. **Studies in the Topography of the Rodent Brain: Erethizon dorsatus and Geomys bursarius.** Herrick (*Bulletin Lab. Denison University*, vol. vi., part 1, June, 1891, pp. 26-46)
10. **Morphology of the Avian Brain.** Turner (*J. of Comp. Neurology* 1. pp. 38-39. pl. v.-viii., also pp. 107-133, pls. xiv., xv., xvi., March, 1891).
11. **Topography and Histology of the Brain of Certain Reptiles.** Herrick (*Journal of Comp. Neurology* I., pp. 14-38, pls. ix. & x., March, 1891).
12. **Contributions to the Comparative Morphology of the Central Nervous System. Topography and Histology**

of the Brain of certain Ganoid Fishes. Herrick (*J. of Comp. Neurology* II. pp. 149-183. pls. x., xi., xii. & xiii., June, 1891).

13. **Cells of the Cerebro-Spinal and Sub-Cutaneous Ganglia in Myxine glutinosa.** Retzius (*Biologische Untersuchungen*, Neue Folge I, mit 13, Abbildungen).
14. **Muscular and Nervous Systems of Dibothriorhynchus benedenii, &c.** Crety (*Bollettino della Societá di Naturalisti di Napoli*, vol. iv. 1890).
15. **Histology of the Nervous System of Hirudineae.** Rohde (*Sitzungsber. der Kgl. Preussischen Akademie der Wissenschaften zu Berlin*, III., 1891).
16. **The Central Nervous System of Cladocera.** Samassa (*Arch. f. Mikrosk. Anat.* xxxviii., 1, pp. 100-142; pls. v., vi., vii.; Sept., 1891).
17. **The Structure of the Nervous Centres in Limulus polyphemus.** Viallanés (*Comptes rendus de l'académie des sciences, Paris*, 1890, Tome cxi.)
18. **The Nervous System of Parmophorus Australis.** Boutan (*Arch. de zoologie exper. et générale.* Série II., Tome 8, No. 2, 1890).
19. **Nervous System of certain Species of Diaptomus.** Richard (*Bul. de la soc. zoologique de France*, vol. xv., No. 10, 1890).
20. **Further Studies on the Brain of Limulus polyphemus.** Packard (*Zool. Anzeiger*, xiv. 361, 1891).
21. **The Nervous System of Crustacea.** Retzius (*Biologische Untersuchungen, Neue Folge*, i. Fourteen plates in folio).
22. **The Origin and Development of the Central Nervous System in Limax maximus.** Henchman (*Bull. of the Museum of Comparative Zoology at Harvard College*, Vol. XX., No. 7, December, 1891. With ten plates).
23. **The Development of the Central Nervous System of Blatta germanica.** Cholodkovisky (*Zool. Anzeiger*, xiv. p. 260, April, 1891).

EMBRYOLOGY.

24. **The development of the Human Hind-Brain, from the end of the first to the beginning of the third month. I. The Medulla Oblongata.** His (*Abh. der Math. Phys. Classe der K. s. Gesellsch. der Wissensch.*, xvii. (xxix.), *Leipsic, Hirzel*, 1891, pp. 74, pls. 4).

A systematic account of the development of the medulla oblongata, illustrated with 18 woodcuts and 4 lithographs. It is impossible to reduce this paper to an abstract.

25. **Appearance of Cells in the Embryonic Cord.** Ramón y Cajál (*Gazeta Sanitaria de Barcelona*, ii. No. 12).

26. **The Myotomes and the Nerves of the back of the Head and Proximal Region of the Trunk in Embryos of Tailless Amphibians.** CHIARUGI (*Arch. ital. de Biol.* xv., ii., pp. 229-291, May, 1891).

MORPHOLOGY.

27. **The Arrangement of the Supra-cerebral Veins in Man, as bearing on Hill's Theory of a Developmental Rotation of the Brain.** BROWNING (*Association of Amer. Naturalists*, September 24th, 1891; *J. of Nervous and Mental Disease* xviii. ii. pp. 711-717. November, 1891).

Evidence in favour of a gliding of the brain and pia upon the dura and brain case derived from the displacement backwards of the posterior supra-cerebral veins.

PHYSIOLOGY: ELECTRIC CURRENTS.

28. **Electro-motor Phenomena in the Brain as expressions of its Activity.** DANILEWSKY (*Centralbl. f. Physiol.*, v. 1, pp. 1-4, April 11, 1891).

Even weak stimulation of a sense organ or of the internal sensory nerve (the Vagus) produces an obvious change in the electro-motor features of the appointed region of the cortex of the brain, usually on the opposite side. Deep morphia or chloroform-narcosis sets it aside. In some cases very complicated negative variations, which last for a couple of seconds, follow assimulation. The electro-motor phenomena of the cortex of one hemisphere can be affected by stimulating the cortex of the opposite hemisphere. The stimulation-processes in the cerebrum which are caused by external stimuli are accompanied by definite electro-motor phenomena.

29. **Mammalian Nervous System: its functions and their Localisation determined by an Electrical Method.** GOTCH and HORSLEY (*Phil. Trans. of the Roy. Soc. of London*, Vol. 182 (October 15, 1891) B. pp. 267-526, plates 29-35).

This magnificent monograph incorporates the substance of the Croonian lecture. It reached us while these abstracts were passing through the press.

30. **The Currents of the Central Nervous System.** CATON (*Centralbl. f. Physiol.*, 1891, pp. 785-786, March 14).

Calling attention to a paper published in the *British Medical Journal*, 1875, ii., p. 278, in which results are recorded similar to those recently published by v. Marxow and Beck. External surface of cortex of cerebrum positive to cut surface. Functional activity accompanied by negative variation.

PHYSIOLOGY.

31. **Phenomena of Movement in the Brain of Leptodora hyalina.** WIEDERSHEIM (*Anat. Anz.*, v. 23, p. 673).

This animal is transparent, and can when narcotised with chloroform be examined for an hour at a time in a condition of immobility.

The œsophageal ganglion or brain properly so called consists of a *pars mobilis* and *partes fixæ*. The former constitutes the anterior third, and consists of a granular mass rich in cells, in which a slow-flowing movement is to be observed, the corpuscular elements presenting changing pictures and vacuoles appearing. The central nerve substance is not fixed in a stiff form, but is capable of active movement.

PSYCHOLOGY.

32. **Guide to Physiological Psychology in Fourteen Lectures.** ZIEHEN (*Jena*, 1891).

33. **Experimental Analysis of Certain Elements of the Sensation of Weight.** CHARPENTIER (*Arch. de Physiol.* iii., 1, p. 122).

CEREBRUM: PHYSIOLOGY.

34. **Ablation of the Cerebral Hemispheres in the Pigeon.** BOECK and BOEUF (*Bull. de la Soc. de Méd. Mentale Belgique*, Dec., 1890).

The animals survived removal of the greater part of the hemispheres for two and a half years. The animal life subsequent to the operation may be divided into three stages. 1. It is very somnolent. 2. It begins to see, but does not feed itself, and is very irritable; in this stage many animals die. 3. The animal gains in weight, drinks when its beak is placed in water. In this stable stage it shows no want of motility, no inco-ordination or loss of balance. It gradually learns to feed itself and to drink, sees and understands what it sees, hears, feels, sleeps at night, chases other pigeons but does not regain sexual instincts.

CEREBRUM: ANOMALY.

35. **Human Cerebrum with a Remarkably Modified Fronto-Parietal Lobe.** TURNER (*J. of A. and P.*, xxv., pp. 327-337).

Fossa of Sylvius open; absence of the Rolandic and præcentral fissures with the transverse convolutions; convolutions antero-posterior, arranged in tiers, having an arcuate disposition in relation to the Sylvan fissure.

ABNORMALITY.

36. **Anatomical Observations on the Brain and Sense-Organs of the Blind Deaf-mute, Laura Bridgman.** DONALDSON (*American Journal of Psychology*, October, 1890).

FORE-BRAIN.

37. **The Relation of the Cerebral to the Olfactory Portion of the Brain.** WILDER (*Assoc. Amer. Anat. Records*, 1890).

CEREBRUM CONVOLUTIONS.

38. **The Sub-frontal Gyre in Man and Apes.** WILDER (*Buffalo Med. and Surgical Journal*, May, 1890).

CEREBRUM CORTEX.

39. **The Cortex of the Cerebrum and Cerebellum.** TARGOWLA

(*Bul. de la societé anatomique de Paris*, V., 6, 1891, pp. 175-176).

40. **Sensibility of the Cerebral Cortex to Chemical Excitation.** GALLERANI AND LUSSANA (*Arch. ital. de Biol.* xv. 3, pp. 396-403, August, 1891).

CORTEX: PHYSIOLOGY.

41. **The Modifications in Excitability of the Cortex induced by Cocain and the nature of the Psychomotor Centres.** BELMONDO (*Lo Sperimentale*, August, 1890).

Cocain only depresses the irritability of the cortex to a certain extent, and B. concludes that it paralyses the sensory elements in the cortex and not the motor.

CORTEX: HISTOLOGY.

42. **Histology of the Cerebral Convolutions of Lower Mammals.** RAMÓN Y CAJÁL (*Gazeta Sanit. Municipal. de Barcelona*, December 15, 1890).

43. **Special Nerve-Cells in the first layer of the Cerebral Convolutions.** RAMÓN Y CAJÁL (*Gazeta Medica Catal., Barcelona*, 1890, vol. xiii.)

CORTEX: CONVOLUTIONS.

44. **Sylvian fissure and island of Reil in the Genus Hylobates.** WALDEYER (*Sitzb. d. kgl. preussischen Akademie der Wissenschaft zu Berlin*, xvi., 1891, pp. 265-267).

CEREBRUM: CONVOLUTIONS.

45. **Origin of the Cerebral Convolutions** (*Continued*). JELGERSMA (*Centralblatt für Nervenheilkunde und Psychiatrie*, XIV., January, 1891).

LOCALIZATION.

46. **Report of Six Lectures on Cerebral Localization, delivered before the Boston Medico-Psychological Society, February and March, 1891.** DONALDSON (From notes by T. L. Bolton, *American Jour. Psychology*, iv. 1, April, 1891).

CORTEX: LOCALISATION.

47. **The Localisation of Taste in the Cortex.** SCHTSCHERBACK (*Centralbl. f. Physiol.* v. 2, pp. 289-298, August 29, 1891).

Gad had concluded that biting, chewing, and swallowing are in the rabbit reflex acts dependent upon the brain-stem, while the formation of the bolus and its backward movement are governed by the cortex. S. attempts to solve two problems: 1. Can these actions be rendered impossible by destruction of any definite part of the cortex? 2. What is the mechanism of the origin of these movements? In all cases in which the power of swallowing at Will is lost, loss of taste is also evident. Injury to an area extending for 2 to 3 mm. in front and behind the line of the coronal suture and vertically from the longitudinal fissure to the lower edge of the brain causes loss of taste of the opposite side of the tongue. The loss of taste lasts for six days at the longest.

LOCALIZATION: THERMOTAXIC.

48. **Heat-Centres.** BACULO (*Istituto. di Patologico gen. dell' Univ. Napoli*, 1890).

49. **Position and Value of those Lesions of the Brain which cause a rise in Temperature.** WHITE (*J. of Physiol. XII.* 3, pp. 271-278, plate vi., August, 1891).

Further observations regarding the effects upon the body-temperature of limited injuries to several parts of the brain. Injury by a blunt probe. When this is passed into the optic thalamus there is an average rise of rectal temperature of 1° F. When it is passed into the corpus striatum the average rise is 1.9° F. If 103° F. be regarded as the highest normal temperature the max. rise above this is slight (.4° F.) when the optic thalamus is injured, as compared with the rise (1.8° F.) when the corpus striatum is injured.

Injury to the septum pellucidum causes a marked rise of temperature. Injury to the cerebellum causes no rise of temperature. Injury to the posterior part of the cerebral cortex produces a more marked effect than injury to the frontal portion. Lesions of the crus cerebri cause a marked rise.

HIPPOCAMPUS.

50. **Histology of the Hippocampus Major.** SALA (*Zeit. f. wiss. Zool.*, Bd. 52, 1891, Heft 1, pp. 18-45).

51. **Histology of the Fascia Dentata Tarini.** SALA (*Verh. Xten intern. Kongresses zu Berlin*, ii. 1, 1890, pp. 153-154).

CORPUS CALLOSUM.

52. **The development of the Corpus Callosum of the Human Brain.** MARCHAND (*Arch. f. Mikrosk., Anat.*, xxxvii., pp. 298 to 334, 2 plates).

The corpus callosum makes its appearance during the fourth month as a round swelling (1 to 1.5 mm. thick) at the upper end of the lamina terminalis, precisely in front of the foramen of Monro. From this spot the corpus callosum extends progressively backwards. The inner arcuate convolution has by the end of the fifth month the appearance of the fornix. The outer arcuate convolution constitutes the fascia dentata. At the sixth month the corpus callosum is 14 mm. long; genu and rostrum are complete. The relation of the corpus callosum to the lamina terminalis and the prolonged lamina terminalis, which forms the floor of the ventricle of the septum, is described.

53. **Development and Minute Anatomy of the Corpus Callosum.** BLUMENAU (*Arch. f. Mikrosk. Anat.* xxxvii., pp. 1 to 15, 1 plate).

Sagittal sections of embryonic human brains show that the corpus callosum originates within the upper arcuate convolution, not between the upper and lower. Its central portion develops first. The histological investigation was carried out in pig-embryos for the most part. When first formed the upper layer of the corpus callosum contains all the elements which occur in the mesial wall of the hemisphere. Its under surface, especially where it bounds the ventriculus septi pellucidi and lies between the pillars of the fornix, is covered with the grey substance of the upper arcuate convolution. The cortex which covers its upper surface contains fibres which connect the tuber olfactorium with the fascia dentata; its grey substance is continuous with the fasciola cinerea; it contains large well-developed cells.

54. **Development of the Corpus Callosum in the Human Brain.** MARCHAND (*Archiv. f. Mik. Anatomie*, Band 37, 1891, Heft 2).

55. **On the Brains of a Sheep and Cat lacking the Callosum.** WILDER (*Assoc. Amer. Anat., Records*, 1890).

56. **Recent Investigations on the Structure and Relations of the Optic Thalami.** RUSSELL PEMBERTON (*J. of Comp. Neurology*, iii. pp. 135-149, June, 1891).

OPTIC THALAMUS.

57. **The Relation of the Thalamus to the Paracœle, especially in the Apes.** WILDER (*Assoc. of Amer. Anatomists, Records*, 1889).

EPIPHYSIS.

58. **The development of the Epiphysis Cerebri.** HECKSHER (*Copenhagen, Reitzel,* 1890, pp. 82, plates 2, 23 lithographed figures).

Exhaustive account of the literature. Proper observations of the embryos of representatives of each class of vertebrates. Relation of the brain outgrowth to the epidermis especially studied.

CEREBRAL PEDUNCLES.

59. **The Course of the Fibres in the Pes Pedunculi and the Cortical connections of the Corpus geniculatum internum.** ZACHER (*Arch. f. Psych.* xxii., p. 654).

CEREBELLUM.

60. **The Cerebellum and its Functions.** COURMONT (*Paris, Alcan,* 1891, pp. 600).

Material; comparative, pathological, experimental. Collected from many different sources with a view to proving that the cerebellum is an *organe de sensibilité* and endowed with psychic functions.

CEREBELLUM.

61. 1. **Illustration of the Architecture of the Cerebellum.** HERRICK (*J. of Comp. Neurology*, i. pp. 5-14, plates i.-iv., March, 1891).

62. **The Connections of the Cerebellum.** BROSSET (*Lyons*, December, 1890).

63. **Origin and Course of the Cerebellar Peduncles and their Relation to the Rest of the Central Nervous System.** MARCHI (*Florence. Le Monnier.*, 5 plates).

Observations upon fifteen animals in some of which one hemisphere, in others the middle lobe, in others the whole cerebellum was removed.

Method: large pieces four to five days in Müller's fluid, divided into pieces of one cm., which are placed for eight days more in the same fluid, and then in an "osmio-bichromic" mixture (two parts Müller's fluid to one part one per cent. solution of osmic acid), in which they lie until hard enough to make into sections. The unaltered fibres are coloured a clear grey or ash colour, the degenerated medullary sheaths, dark grey or black.

Amongst Marchi's conclusions are the following:—The brachia conjunctiva do not cross completely, but give some fibres to the optic thalamus of the same side; most of their fibres go to Stilling's nucleus of the opposite side. No fibres go either to the optic tract or to the bundle of Reil. The middle peduncles are not solely commissural, but contain fibres which join the grey matter on both sides. The fibres in the posterior peduncle for the opposite olive are both afferent and efferent. The posterior longitudinal bundle and the fillet have a common origin in the cerebellum, chiefly in the middle lobe, they connect the nuclei of the cranial nerves with the cerebellum. The fillet connects the cerebellum with the pons, corpora quadrigemina, and probably with the corpus striatum. The posterior longitudinal bundle joins the fillet about the level of the olive, and both go into the antero-lateral tracts, and probably are thus connected directly with the anterior horn and indirectly with anterior roots. The origin of all these peduncles is spread over the whole grey matter, but the brachia are connected chiefly with the nucleus dentatus, the middle peduncles with the vermis.

64. **The Fossa Vermiana in Primates.** MORSELLI (*Archivio di psichiatria*, Vol. VI., Fasc. iii., iv., 1890).

CEREBELLUM: HISTOLOGY.

65. **Certain Bi-polar Elements of the Cerebellum and new facts with regard to the Evolution of the Cerebellar Fibres.** RAMÓN Y CAJÁL (*Intern. Monatsschr* ii., pp. 445-468, 1890).

Fœtal mammalian brains investigated with Golgi's method. The nuclei which in the fœtal or new-born brain lie immediately beneath the pia are divisible into two layers. The superficial layer consists of spherical epitheloid cells with a short process directed towards the surface; the deep layer consists of bi-polar cells, the long axes of which are placed parallel to the surface; the finer of the two processes is to be regarded as the axis-cylinder, it appears to end freely. Peculiar spindle-shaped cells, the long axes of which are vertical to the surface, are to be found in the molecular layer, their axis-cylinder processes pass outwards and join the fibres which lie in the deeper part of the superficial nuclear layer almost at right angles. Two stages are traced in the formation of the cells of Purkinje. 1st, the formation and ramification of their protoplasmic processes; 2nd, their reabsorption or retreat. The fibres connected with each cell of Purkinje form a plexus around its main ascending branch "*l'aborisation ou plexus grimpant.*"

MEDULLA OBLONGATA.

66. **The Outer Nucleus of the Funiculus Cuneatus in the Medulla Oblongata.** BLUMENAU (*Neurol. Centralbl.*, x., 8, April 15, 1891).

Lockhart Clarke described "inner and outer restiform nuclei." The division is usually overlooked or the outer nucleus is described as inconstant (Obersteiner). B. investigated the topography of this nucleus in adult and embryonic brains. It appears at the level in the medulla oblongata at which arcuate fibres begin to pass from the direct lateral cerebellar tract backwards to the inferior end of the restiform body. Only the inner nucleus is to be found lower in the medulla, but even this presents isolated cells which, by their size and form, are judged to belong to the outer nucleus. At first scattered about, they then pass to the periphery, and collect into little groups which lie between the nucleus funiculi gracilis and nucleus funiculi cuneatus or between the latter and the substantia Rolandi. Similar cells are to be observed in the funiculus gracilis, especially in its outer part. The division between inner and outer nuclei is never complete. At first the cells of the outer nucleus are separated by fibres, but afterwards the nucleus becomes very uniform. The cells are multipolar, vesicular, and contain a well-developed nucleus with a nucleolus. Their long diameter equals 50-80 μ, while the cells of the inner nucleus are not usually more than 25·40 μ in diameter. They have a notable resemblance to the cells of Clarke's column. In anatomical situation, too, they seem to belong to this column which elsewhere has a tendency to throw off a detached piece into the midst of the posterior column. The outer nucleus has a considerable size at levels from which the inner has disappeared. On the lateral and ventral sides the nucleus is bounded by a half ring of fibres of the cuneate fascicle which separate it from the restiform body. Ventralwards a layer of grey matter of varying thickness sinks between the cuneate fascicle and the ascending trigeminal root. The upper end of the nucleus reaches to the point of exit of the most posterior auditory fibres. Then follows an account of the several views held as to the destination of the fibres of the cuneate fasciculus. B. believes that many sets of fibres connect the nuclei of the posterior column with the restiform body. He was unable to determine whether the fibres originate in the large cells, but saw the axis-cylinder processes of the large cells of the inner cuneate nucleus passing towards the outer margin of the fasciculus.

SPINAL CORD : DEVELOPMENT.

67. **The Development of the Posterior Fissure and of the Central Canal of the Spinal Cord.** ROBINSON (*Studies in Anatomy from the Anat. Depart. of Owens College*, i. pp. 67-103, pls. ii., iii., 1891).

Observations upon rats and mice. Careful measurements of size at different periods. In the earlier stages the canal increases in size simultaneously with the growth of the other constituents of the cord. After this it diminishes in dorso-ventral diameter; as this diminution commences before the posterior columns have overlaid the cord, it is not due to their pressure; nor is it due to fusion of the lateral walls of the canal. The conclusion of His that an axis-cylinder is the outgrown process of a nerve-cell, and extends from its parent-cell to the end organ or to some other cell confirmed. The posterior columns consist, at least in the early stages, principally of posterior root-fibres. There is no posterior fissure in the proper sense of the term; the tissue lying between the posterior columns is a modified portion of the original cellular wall of the central canal.

SPINAL CORD : COMPARATIVE.

68. **The arrangement of Fibres in the Spinal Cord.** SCHAFFER (*Arch. f. Mikrosk. Anat.* xxxviii., pp. 157-176, plate ix., Sept., 1891).

Investigation of the arrangement of the fibres in blind-worm, grass-snake, tortoise, and lizard; rabbit, bat, and cat. Among other results Schaffer proves that fibres from both sides of the posterior horn go directly through the anterior commissure into the opposite anterior column. Fibres also go from the lateral column into the opposite anterior column. In the snake, fibres from the mesial side of the posterior root pass directly into the anterior column of the opposite side, but it is not certain whether the connection with the root is direct in the blind-worm.

SPINAL CORD : PHYSIOLOGY.

69. **Symptomatology of Total Transverse Lesions of the Spinal Cord.** BASTIAN (*Medico-Chir. Trans.*, lxxiii., pp. 151-217, 1890).

This paper was omitted from the *Abstracts* at the time of its publication as treating of the pathology of the nervous system.

It should, however, find a place in the record of the anatomy and physiology of the system. It is a protest against considering that it is proved for Man that total transverse lesion of the cord is followed by exaggeration of the reflexes in the region below the lesion, after the effects of shock have passed off.

SPINAL CORD: CONDUCTION.

70. **Afferent Routes in Spinal Cord studied by Degeneration.** ODDI AND ROSSI (*Arch. ital. de Biol.*, xv., ii., p. 203, May, 1891).

SPINAL CORD: NERVE ROOTS.

71. **The influence of the Posterior Roots upon the excitability of the Anterior ones.** BELMODO AND ODDI (*Rivista sperimentali di Freniatria*, xvi., 3).

When the posterior roots are cut or paralysed by cocain, the irritability of the motor roots falls. After section of the posterior roots a short stage of increased irritability follows, due to the effect of the section. In the same way stimulation of the central stump prevents the diminution of irritability. This influence is not affected by cutting across the medulla oblongata. The observers, therefore, conclude that stimuli are constantly reaching the central nervous system from the periphery and streaming across into the motor roots.

SPINAL CORD: HISTOLOGY.

72. **The Histology of the Neurogleia and Nerve-substance of the Spinal Cord.** LAVDOWSKY (*Arch. f. Mikrosk. Anat.*, xxxviii., 2, pp. 264-301, plates xiv.-xviii., Oct., 1891).

SPINAL CORD: HISTOLOGY.

73. **The collateral Fibres of the White Substance of the Cord of Batrachian Larvæ.** RAMÓN Y CAJÁL (*Gazeta Sanataria de Barcelona*, October 10, 1890.

SPINAL CORD: MENINGES.

74. **Connection of the Spinal Cord with the Pia Mater.** ASPLUND (*Nordiskt Medicenskt Arkiv.* XXII., Nov. 4, 1890).

CRANIAL NERVES.

75. **The First Appearance of the Cranial Nerves in the Cat.** MARTIN (*Oesterreichische Monatschrift für Tierheilkunde*, xv., September, 1890.

CRANIAL NERVES: I.

76. **The first development of the Olfactory Nerves.** KÖLLIKER (*Sitzungsb. d. phys. med. Ges. zu Würzburg*, 8 and 9, 1890).

The olfactory nerves do not, as hitherto believed, originate from the olfactory bulb, but (as taught by His) they arise in the epithelium of the olfactory region, run centripetally in the direction of the olfactory lobe, and form a secondary connection with this. The fibres of the olfactory nerve are equivalent to groups of nerve cells, and those fibres which possess a number of nuclei are a complex of nerve cells.

77. **First Phases of development of the Cranial Nerves in Mammals and in particular of the Olfactory Nerve.** Chiarugi (*Arch. ital. de Biol.* xv. 3, pp. 418-464, August, 1891; also *Monitore Zool. ital.* ii. 3, with plates).

C. concludes that it is useless to look for the primitive origin of the cephalic and spinal ganglia in the "intermediate groove and ridge" (Zwischenrinne und Zwischenstrang) of *His*, although this fold certainly exists. It is not due to artificial folding. *Contra His* the olfactive and auditory fossæ cannot be derived from the intermediate groove. On the other hand C. does not agree with Beard that the cephalic ganglia arise from the ectoderm independently of the medullary fold, but thinks that they proliferate from its edges. C. describes the olfactory nerve as originating with a conical base from the ventral side of the wall of the forebrain before the appearance of any trace of an olfactory lobe. After a short course it applies itself to the olfactory fossa. It is purely cellular and may be properly called the "olfactory ganglion." In this, again, C. differs from *His* who believes that the olfactory ganglion develops in immediate connection with and at the expense of the olfactory plaque, being therefore at a certain stage unconnected with the brain.

78. **The Olfactory Lobes of Fulmarus glacialis.** Klinckowstrom (*Biologiska Foreninges Forhindingar*, iii. 1, 2, Stockholm, 1890-91).

79. **The partial occlusion of the Olfactory lobe in the Canidae** Fish (*Amer. Monthly Microsc. J.*, xii. 3, pp. 49-52, plate, March, 1891).

The olfactory ventricle is closed by an ingrowth of neurogleia. In some wild as well as in domesticated dogs the cavity of the crus is completely closed, the bulb only partially so. Acuteness of smell cannot be therefore essentially dependent upon the relative size of the rhinocoele.

80. **Origin and Termination of the Olfactory Nervous Fibres.** Ramón y Cajál (*Gazeta Sanit. Municipal. de Barcelona*).

Cranial Nerves: II.

81. **The Course of the Fibres in the Optic Nerves.** Hebold (*Neurol. Centralbl.* x. 6, pp. 167-169, March 15, 1891).

Conclusion from pathological observations, that in each optic nerve fibres of the tract of both sides course in closed bundles. The uncrossed bundle lies throughout on the outer margin of the optic nerve, chiasma and tract. The crossed bundle is central in the nerve, ventral in the tract.

82. **Vestiges of Epithelium on the Optic Nerve and on the Retina.** Ucke (*Arch. f. Mikrosk. Anat.*, xxxviii., 1, pp. 24-38, plate ii. September, 1891).

The retinal epithelium is usually described as ending at the point of entrance of the optic nerve. Ucke shows that it is to be recognised around the optic nerve and also in the optic cups "for a long time." The cavity of the optic nerve is displaced dorsalwards.

83. **The Nervous Elements in the Human Retina.** DOGIEL (*Arch. f. Mikrosk. Anat.*, xxxviii. 3, pp. 317-345, plates xix.-xxii., October, 1891.

A continuation of the author's work upon the retina of the submammals, showing that the same conditions obtain in man.

CRANIAL NERVES: VII.

84. **The Geniculate Ganglion, its Nerves and their Connections.** PENZO (*Ricerche Anatomiche, Atti del Reale Istituto Veneto*, Tomo xxxviii., Series 7, Dispensa 2, 1890-91).

CRANIAL NERVES: IX., X. & XI.

85. **Origin of the ninth, tenth, and eleventh Cranial Nerves.** KOCH (*Nordiskt mediciniskt arkiv.*, xii., No. 11, 1890).

CRANIAL NERVES: X.

86. **Communicating Branch between Superior and Inferior Laryngeal Nerves.** HOWELL and HUBER (*J. of Physiol.*, xii. 1, pp. 5-12, April, 1891).

In the dog the branch was proved by experiment to be a sensory branch of the superior laryngeal distributed to the trachea and œsophagus, although fused in part of its course with the inferior laryngeal.

SPINAL GANGLIA.

87. **The Spinal Ganglia.** MULLER (*Biolog. foren. forh.*, Bd. 2, 1890).

REFLEX.

88. **New Contribution to the Knowledge of the Reflexes.** ERBEN (*Wiener Med. Wochenshr.*, 1890, p. 879).

E. calls attention to the fact that two-thirds of the large cells of the anterior horn may be destroyed and yet the tendon reflexes increased, not depressed. That exaggeration of the deep reflexes on the non-paralysed side is almost always observable where old hemiplegic lesions with secondary degeneration are present. On the non-paralysed side contractures are also apt to occur. The variable effects of cerebral hæmorrhage upon tendon reflexes, explained as depending upon whether the injury secondarily affects the medulla and spinal cord, especially as regards its blood supply.

REFLEX: ANAL.

89. **The Anal Reflex: its Physiology and Pathology.** ROSSOLIMO (*Neurol. Centralbl.*, x., 9, pp. 257-259, May 1, 1891).

Localised in the cord of dogs as the fourth pair of sacral roots and up to the level of the third pair in the cord, lower than any skin reflex even that of the soles.

PERIPHERAL NERVES.

90. **Impossibility of Tiring the Nerves of Mammals.** BOWDITCH (*Du Bois Reymond's Arch.*, 1890, p. 505).

Sciatic nerve stimulated and contraction of tibial muscle recorded. Nerve endings paralysed with curari; continued stimulation of the nerve; as the effects of the curari pass off the contractions of the muscle are resumed.

91. **The Limb Plexuses of Mammals.** PATERSON (*Studies in Anatomy from the Anat. Depart. of Owens College*, i. pp. 135-159, 1891. Reprint from *J. of Anat. and Physiol.*, 1887).

92. **Morphology of the Sacral Plexus in Man.** PATERSON (*Studies in Anatomy from the Anat. Depart. of Owens College*, i. pp. 159-167, 1891. Reprint from *J. of Anat. and Physiol.*, 1887.)

93. **Segmental Distribution of Sensory Nerves.** ROSS (*Studies, in Anatomy from the Anat. Depart. of Owens College*, i. pp. 167-197, 1891. Reprint from BRAIN, 1888).

SENSES: SEMI-CIRCULAR CANALS.

94. **Physiology of the Internal Ear.** FANO AND MASINI (*Centralbt. f. Phys.*, 1891, p. 787, March 14).

A number of experiments upon doves. Disturbance of movement does not accord with the direction in space of the canal which is cut. Permanent disturbance follows section of the horizontal canal only. The movements of the head which follow extirpation of a canal depend upon auditory vertigo, and are not due to irritation-processes, but to an abolished function; they cease after destruction of the membranous cochlea. Doves in which both cochlea and canals are destroyed react to sound. Destruction of the cochlea produces disorders of movement, but they are not permanent, and it is quite clear that the canals serve fairly well for audition. Removal of the cochlea does not diminish the forced movements which follow removal of the cerebellum.

SYMPATHETIC SYSTEM.

95. **The Relation of the Nerve Fibres of the Cervical Sympathetic to the Ganglion Cells of the Upper Cervical Ganglion.** LANGENDORFF (*Centralbl. f. Physiol.*, v., 5, pp. 129-131, June 6, 1891).

Certain observations confirming the conclusion of Langley and Dickinson that nicotin poisons the ganglion cells in which the fibres are interrupted before it poisons the fibres. When an animal is killed by bleeding or suffocation, the action of the sympathetic upon the eye can be shown by stimulating the fibres above the ganglion, when the fibres below the ganglion are inactive, owing to the impassable barrier interposed by the dead ganglion cells.

96. **Functional relations of the Cervical Sympathetic with the Epidermis and Glands.** ARLOING (*Arch. de Phys.*, i., p. 160).

Histological changes in the skin and glands of the tip of the nose of the dog and the muzzle of the ox after section of the cervical sympathetic.

97. **The Relation Between the Sympathicus and Head-Ornament of Certain Birds.** JEGOROW (*Du Bois Reymond's Archiv.*, 1890, Suppl., p. 33).

The anatomy and physiology of the vaso-motor nerves of the head ornaments of the turkey and other fowls in which these decorations are capable of erection under the influence of psychical stimuli.

98. **The Sympathetic of Elasmobranch and Osseous Fishes.** CHEVREL (*Thèse. Poitiers, Oudin*, 1890, 8vo, pp. 203).

98* **The Functional Relations of the Sympathetic Chain with Skin and Glands.** ARLOING (*Verhandl. des Xten internat. medic. Kongresses zu Berlin*, 1890, Bd. ii. 2, pp. 9-11; also *J. de Med. veterinaire et Zootechnique, Lyons*, xv. pp. 617-627, 1890).

PILO-MOTOR.

99. **Pilo-motor Nerves.** LANGLEY and SHERRINGTON (*J. of Physiology* xii., 3, pp. 278-292, August, 1891).

The hairs of the monkey, cat and probably all animals in which horripilation occurs under the influence of strong emotion, may be erected by stimulation of nerve-fibres which issue from the spinal cord and pass through the sympathetic nervous system. The effects are mainly unilateral, but in the tail they are bilateral.

GLANDULAR.

99* **Course and Connections of Secretory Fibres Supplying the Sweat Glands of the Feet of the Cat.** LANGLEY (*J. of Physiology*, xii., 4, pp. 347-375, pls. xii. and xiii., September, 1891).

100. **Connection with Nerve Cells of Vaso-motor Nerves for the Feet.** LANGLEY (ditto, pp. 375-378). .

BLADDER.

101. **The Motor Nerves of the Bladder.** NAWROCKI and SKABITSCHEWSKY (*Arch. f. Physiol.*, xlviii., 7 and 8, p. 335).

The bladder is supplied by two sets of motor nerves. 1. The upper through the ganglion mesentericum inferius. 2. The sacral nerves within the pelvis. The former come from the spinal cord *via* the fourth and fifth anterior lumbar roots, rami communicantes, lumbar part of the sympathetic chain and nervi mesenterici; the latter by the second and third sacral roots.

SUPRARENAL.

102. **The Termination of Nerve-fibres in the Suprarenal Capsule of Mammals.** FUSARI (*Atti. della R. Accademia delle scienze di Torino*, Vol. XXVI., Dispen. 5, 1890-91).

VASO-MOTOR.

103. **The Vaso-motor Nerves of the Retina and Particularly the Trigeminus.** DOYON (*Arch. de Physiol.*, iii., 3, p. 13).

Dilation of the vessels of the retina on stimulation of the gasserian ganglion.

104. **The Motor Nerves of the Portal Vein.** MALL (*Du Bois Reymond's Archiv.*, 1890, Suppl., p. 57).

105. **The Vasomotor Function of the Spinal Cord.** ADUCCO (*Arch. ital. de Biol.*, xiv., 3, p. 373).

106. **Intracranial Course of the Auricular Vasomotor Nerves.** MORAT (*Arch. de Physiol.*, iii., 1, p. 87).

TROPHIC NERVES.

107. **Trophic Nerves.** ARND (*Archiv. f. Anatomie u. Physiologie, Physiologische Abtheilung*, 1891, p. 54).

HISTOLOGY.

108. **The Structure of the Nerve Cells in the Sympathetic of Amphibia.** SMIRNOW (*Arch. f. Mikr.*, xxxv., p. 407).

Injection of methyl-blue *intra vitam.* The network which invests the cells is a genuine closed network with varicose fibrils. The spinal fibre originates in the network and acquires a myelin sheath sometimes at a considerable distance from the cell. The cell-body and its straight fibre stain badly with methyl-blue. The spinal fibre courses peripherally, and contains fibres which enter into connection with various peripheral structures.

109. **The Diffuse Nerve-Network of the Nerve Centres.** GOLGI (*Arch. ital. de Biol.*, xv., iii., pp. 434-463, August, 1891).

A consideration of the ultimate constitution of grey matter in its anatomical, physiological and psychological aspects.

HISTOGENY.

110. **Development of the Ganglion-rudiments in the Human Embryo.** LENHOSSÉK (*Arch. f. Anat. u. Phys.*, Anat. Abtheilung, 1891, p. 1).

HISTOLOGY: FIBRES.

111. **The Optical Relations of Medullated and non-Medullated Nerves.** AMBRONN (*Berichte der königl. sächs Gesellsch. der Wissensch.*, p. 421, 1890).

Myelin.

112. **Medullated and non-Medullated Nerves.** Gad and Heymans (*Archiv f. Anat. und Physiol.*, p. 530, 1890).

An attempt to define myelin chemically. Which is it of all the chemical substances separated from nerves up to the present? Regarding myelin as the substance which gives the "myelin-formation" with water and blackens with osmic acid, the authors conclude that myelin is lecithin in a free state or in loose chemical combination. They distinguish nerve-fibres with myelin-containing medullary sheaths, with or without Schwann's sheath; nerve-fibres with myelin-free medullary sheaths with or without Schwann's sheath, and free axis-cylinders. Except in the lower vertebrates the roots of the spinal nerves contain no myelin-free fibres. It is questionable whether the statement holds good that all myelin-containing nerve fibres which originate in the cerebro-spinal system lose their myelin by passing through sympathetic ganglia.

113. **The termination of Nerves in the Mucous Membrane and Serous Glands of the Tongue of Mammals.** Fusari and Panasci (*Arch. ital. de Biol.*, xiv. 3, p. 240, 1 plate).

114. **The Annular Bands of the Nerve-fibre.** Johansson (*Centralbl. f. Physiol.*, v. 11, pp. 299-301, August 29, 1891).

115. **Certain peculiarities of structure of Medullated Nerves.** Marenghi and Villa (*Arch. ital. de Biol.*, xv. 3, pp. 404-408, August, 1891).

116. **The structure of the Nerve-fibre.** Owsjanikow (*Bull. de l'Acad. imp. des Sciences de St. Petersburg*, i. 4, p. 497).

The effects of treatment with silver-nitrate studied. Nitrate of silver gives rise to artificial products. The transverse striation is limited to the outer surface of the axis-cylinder, and does not affect the primitive fibrillæ. The keratin-network of Ewald and Kühne is not an artifact.

Nerve-endings.

117. **The Terminal Nerve-plaques in the Tendons of Vertebrates.** Ciaccio (*Arch. ital. de Biol.*, xiv. 1 & 2, p. 31).

Pathology.

118. **Paralytic Idiocy in Dogs.** Gerdes (*Inaug. diss. Berlin*, 1891).

Condition similar to dementia paralytica both in symptoms and histological changes induced by repeated rotations.

Circulation.

119. **Relation of the Arteria Choroidea Anterior to the Posterior Limb of the Internal Capsule.** Kolisko (*Wien* 1891. *Hölder*).

CIRCULATION: CEREBRUM.

120. **Cerebral Circulation.** BOECK AND VERHOOGEN (*Brussels*, 1890).

1. In asphyxia the amount of blood circulating in the brain is everywhere increased owing to the active dilation of its vessels. 2. Injection of sulphuric ether raises the general blood pressure, but increases the amount of blood circulating in the brain in still greater proportion. 3. Intravenous injection of morphia renders the cortex anæmic and the base of the brain hyperæmic.

121. **Influence of Attitude on Cerebral Anæmia.** RICHET (*C.R. Soc. de Biol.*, Jan. 17th, 1891, p. 35).

If a large artery of a dog is cut while the animal is held in an upright position, respiration ceases in less than half a minute. It is prolonged by lowering the head.

BRAIN-WEIGHT.

122. **The Brain-weight of certain Animals.** MIES (*Verhandlungen der Gesellschaft deutscher Naturforscher und Aerzte*, 63. *Versammlung zu Bremen, September*, 1890, *Leipzig*, 1891).

Intracranial pressure is always positive. It varies in the dog from 72 to 190 mm. of water. It is increased when the intracranial space is diminished by the introduction of foreign bodies. All the ordinary symptoms of pressure upon the brain are produced by introducing a fluid or an india-rubber bladder between the dura mater and the bone. Brain-anæmia ensues when the intracranial pressure is raised. Brain-pressure is not increased by injecting neutral or ammoniacal salt solution into its blood-vessels (*contra* Adamkiewicz). Local alterations in pressure were made by placing pieces of laminaria within the skull. Death is produced when the skull space is curtailed by more than $\frac{1}{19}$ to $\frac{1}{20}$ of its volume (*contra* Adamkiewicz, who sets this figure at $\frac{1}{4}$ to $\frac{1}{6}$).

BRAIN-PRESSURE.

23. **Brain-pressure.** CYBULSKI (*Centralbl. f. Physiol.*, iv., 26, pp. 834-6, March 28, 1891).

METHOD.

124. **Staining of the Axis-cylinder in the Spinal Cord.** SCHMAUS (*Münchener Med. Wochenschr.*, 8, 1891).

Rub up 1 gramme of sodic carminate (Carminsaüres Natron) with $\frac{1}{2}$ gramme nitrate of uranium, boil for half an hour in 100 grammes of water and filter when cold. Sections lie in this solution for 15-20 minutes. Tissues must be hardened in Müller's fluid.

125. Three New Methods for Staining the Medulla and Axis-cylinder by means of Hæmatoxylin. Wolters (*Ztschr. f. Wiss. Mikrosk.*, vii.)

1. Tissues hardened in Müller's fluid dehydrated and embedded in celloidin. Sections placed in a solution of hæmatoxylin 2 parts dissolved in a minimum of absolute alcohol and added to 100 parts 2 per cent. acetic acid. The staining solution is kept at 45° C. The sections are then placed in Müller's fluid and differentiated by the potassic permanganate method. Medullated fibres come out blue black.

2. Sections from tissues hardened in 50 per cent. alcohol saturated with bichromate of potassium and sulphate of copper, dissolved by using 5 or 6 drops of glacial acetic acid to the 100 parts are macerated for 24 hours in a mixture of 2 parts of a 10 per cent. solution of chlorate of vanadium and 8 parts of an 8 per cent. solution of acetate of aluminium; washed in water 10 minutes and placed in the above-mentioned hæmatoxylin solution for 24 hours at 45° C.; placed in 80 per cent. alcohol, acid with hydrochloric acid, until they assume a clear blue-red colour; then washed in weak alcohol. For peripheral nerves this gives a staining of the axis-cylinder. 3. By this latter method the pyramids of the cerebrum are coloured deep blue. Axis-cylinders and gleia-cells are also stained.

126. Impregnation of the Central Nervous System with Quick Silver. Cox (*Weekbl. van het Nederl. Tydschr. v. Geneesk*, ii. 15, 1890).

Modification of Golgi's method.

Chemical Composition.

127. Influence of Chloride of Sodium upon the Chemical Composition of the Brain. Novi (*Arch. ital. de Biol.*, xv. 2, p. 203, May, 1891).

RULES

AND

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AND

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RULES.

1.—The Society shall be called the NEUROLOGICAL SOCIETY OF LONDON.

2.—The objects of the Society shall be to promote the advance of Neurology, and to facilitate intercourse amongst those who cultivate it, whether from a Psychological, Physiological, Anatomical, or Pathological point of view.

3.—The Society shall consist of Ordinary, Corresponding, and Honorary Members.

4.—Any one engaged in researches bearing on Neurology, or manifesting interest in such researches, shall be eligible for the Ordinary Membership.

5.—Men of distinction in Science, who have contributed to the advancement of Neurology, shall be eligible for the Honorary, or for the Corresponding, Membership.

6.—The number of Honorary Members shall be limited to six, and that of Corresponding Members to twelve.

7.—Honorary and Corresponding Members shall have the right of attending the Meetings of the Society, and of taking part in them.

8.—Honorary and Corresponding Members shall be elected by the Society on the recommendation of the Council.

9.—Candidates for Ordinary Membership shall be recommended by at least three members of the Society, who shall append their names to a printed form supplied by the Secretaries to any persons seeking membership.

10.—The Council shall nominate Candidates for election into the Society as Ordinary Members, from among persons so recommended.

11.—The names of Candidates nominated by the Council shall be submitted to the next Meeting of the Society, and shall be ballotted for at the next subsequent Meeting, one black ball in ten excluding.

12.—The Annual Subscription of an Ordinary Member shall be One Pound, due in advance.

13.—The Treasurer shall send to each Member, shortly before the Annual General Meeting, a notice reminding him that his Subscription becomes due on that day.

14.—Non-payment of the Subscription within twelve months after it is due shall be considered as equivalent to resignation.

15.—Absence of any Member residing within the Metropolitan area, from all Meetings held during the year, shall be considered as equivalent to resignation.

16.—The Council shall consist of a President, two Vice-Presidents, two Secretaries, one Treasurer, and ten Councillors.

17.—Five Members of the Council shall form a quorum.

18.—The office of President shall be tenable for one year, and be entered upon at the beginning of each year by the Senior Vice-President.

19.—The office of Vice-President shall be tenable for two years; one Vice-President being elected every year.

20.—The Secretaries and Treasurer shall be elected annually, with eligibility for re-election.

21.—The Councillors shall be elected for one year, and not more than eight shall be eligible for re-election to the same office during the following year.

22.—One full week before the Annual General Meeting, the Secretaries shall send to each Member a ballotting paper containing the names of the Officers and Councillors whom the Council nominate for the ensuing year.

23.—The Society shall elect the Council by ballot, each Member, however, being at liberty to substitute other names for any of those upon the list.

24.—There shall be six ordinary meetings, annually, of which the first held in each year shall be the Annual General Meeting.

25.—Notices of each meeting, and of the subjects to be considered, shall be sent by the Secretaries to each Member of the Society at least one week before the meeting.

26.—Special Meetings shall be held at the option of the Council, or at the request, in writing, of twenty Members.

27.—The President shall have the power of inviting any person to attend and to take part in the scientific work of a meeting.

28.—If at any time the Council shall be of opinion that the interests of the Society require the expulsion of a Member they shall submit the question to a Special General Meeting, at which, if more than one-half of the members of the Society vote, by ballot as usual, for the expulsion of the member his subscription for the current year shall be returned to him and he shall thereupon cease to be a Member of the Society.

29.—The Council shall draw up, and submit for the approval of the Society supplementary rules regulating the dates and places and character of the meetings; shall propose special subjects for investigation by the Society, and shall nominate sub-committees for the methodical carrying out of such investigations. They shall decide, from time to time, on the form of publication which its proceedings are to assume.

30.—No alteration shall be made in the present rules, excepting at the Annual Meeting, or at a Special Meeting, convened for the purpose, and unless it be proposed by the Council, or in writing by at least twenty Members, the usual notice being given of the proposed change to every Member before the meeting at which it is to be brought forward.

Each Member receives quarterly, from the beginning of the year in which he is elected, a copy of " BRAIN; a Journal of Neurology," the organ of the Society, and edited for it by A. de Watteville, M.A., M.D., B.Sc.

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⁂ Members are requested to communicate with the Secretaries when corrections are necessary.

EXPLANATION OF ABBREVIATIONS.

O.M., Original Member.
Pres., President.
V.P., Vice-President.
Tr., Treasurer.
Sec., Secretary.
C., Member of Council.

* * *

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